DOCTOR OF PHILOSOPHY

The modification of attentional bias to emotion-related words using the unilateral hand contraction method

Walsh, Francesca

Award date:
2015

Awarding institution:
Coventry University

THE MODIFICATION OF ATTENTIONAL BIAS TO EMOTION-RELATED WORDS USING THE UNILATERAL HAND CONTRACTION METHOD

by

FRANCESCA A. E. WALSH

A Thesis submitted to

Coventry University

for the degree of

DOCTOR OF PHILOSOPHY

School of Psychological, Social and Behavioural Sciences

Faculty of Health and Life Sciences

Coventry University

September 2015

Abstract

Substantial evidence shows that attentional bias towards threat plays a fundamental role in anxiety and that deficits in frontal brain functioning might explain this. However, a paucity of research on anger related attentional bias leaves unanswered questions about whether similar mechanisms underpin aggression. This has led to a lack of theoretical explanations for anger related attentional bias and effective interventions to reduce anger. Electroencephalographic (EEG) evidence suggests that the hemispheric specialisation of the frontal brain predicts differential responding to emotional stimuli in anger and anxiety. Manipulating motivational direction, via unilateral hand contractions (UHCs), provides a means to explore the causal relationship between anger and attentional bias to threat. Previously, this method has only been used to change experiential and expressional aspects of emotion and its effectiveness in modulating attentional components of emotion regulation are unknown. Therefore, this Thesis aims to explore whether UHCs effectively modulate attentional bias to threat in relation to, and independent of trait anger. It also aims to discover the underlying neural effects of the UHC method to examine whether threat-related attentional changes reflect modulations in cognitive control and/or approach motivation. Finally, this Thesis aims to bridge the gap between the attentional bias and frontal brain asymmetry literature. These aims will be addressed by employing Emotional Stroop and Dot Probe paradigms as well as event related potentials measures. The findings provide evidence that UHCs provides an effective technique to modulate attentional bias to threat. Specifically, RHCs reduce attentional bias to threat independent of trait anger and in individuals with low trait anger but they do not modify attentional bias to threat in high anger individuals. In contrast, LHCs increase attentional bias to threat and this reduced task relevant processing, independent of trait anger. The implications of these novel findings and future directions of research are discussed.

Dedication

For my children, with love. Never let life's hurdles stop you achieving your dreams. Listen to your dreams, allow yourself to believe that anything is possible, then make it happen.

Acknowledgements

I would like to take this opportunity to acknowledge a number of people who made the creation of this Thesis possible.

Firstly, I would like to thank all of the people who dedicated their time to participate in the current research.

I would also like to thank Anthony McGuffie for his time, patience and assistance in writing presentation programmes used in the research in this Thesis.

I am also eternally grateful to Sarah, Mike, and Jayne for their continual support, encouragement and friendship that spurred me on during the PhD process.

Special thanks also goes to my three beautiful children, Natalie, Dillon and Ashleigh for their patience, understanding and love during times where my presence as a mother has been somewhat overshadowed by the PhD. I would also like to thank Ashleigh for a continual supply of coffee and food that kept me going when tiredness took hold.

Finally I would like to express my sincere gratitude to Dr John Williams, and Professor Jochen Kaiser, who have been a valued source of advice. I would also like to give a heartfelt thanks to Dr Rebecca Jenks who in the final months of the PhD was my rock, going above and beyond her role as a supervisor to ensure my sanity remained intact and the smooth completion of this Thesis. Thank you for your never ending inspiration and encouragement.

This research was funded by a Coventry University studentship.

List of Contents

List of Illustrations

List of Tables

List of Abbreviations

ABM	Attentional Bias Modification
ACC	Anterior Cingulate Cortex
ACT	Attentional Control Theory
ADM	Affective Decision Mechanism
ANOVA	Analysis Of Variance
AX-I	Anger Expression-In
AX-O	Anger Expression-Out
BAS	Behavioural Activation System
BESA	Brain Electric Source Analysis
BIS	Behavioural Inhibition System
DLPFC	Dorsolateral Prefrontal Cortex
DPT	Dot Probe Task
EHI-r	Edinburgh Handedness Inventory-revised
EEG	Electroencephalogram
EOG	Electro-occulargram
EST	Emotional Stroop Task
ERPs	Event Related Potentials
fMRI	Functional Magnetic Resonance Imaging
GAM	General Aggression Model
GES	Goal Engagement System
GTES	Guided Threat Evaluation System
Hz	Hertz
LHCs	Left Hand Contractions
LQ	Laterality Quotient
µV	Microvolt
NAS	Negative Affect Syndrome
NSW	Negative Slow Wave
NHCs	No Hand Contractions
OFC	Orbitofrontal Cortex
SIP	Social Information Processing
STAXI-2	State Trait Anger Expression Inventory
RAS	Resource Allocation System
RHCs	Right Hand Contractions

RT	Reaction Time
rTMS	Repetitive Transcranial Magnetic Stimulation
PDP	Parallel Distributed Processing
ηp2	Partial eta squared
PET	Positron Emission Tomography
PFC	Prefrontal Cortex
PTES	Pre-attentive Threat Evaluation System
SNR	Signal to Noise Ratio
UHCs	Unilateral Hand Contractions
VES	Valence Evaluation System
vmPFC	Ventromedial Prefrontal Cortex

CHAPTER 1

ANGER-RELATED ATTENTIONAL BIAS TO THREAT AND FRONTAL BRAIN ASYMMETRY

1. Introduction

A wealth of evidence exists to show that attentional bias towards threat plays a fundamental role in the aetiology and maintenance of negative affect such as anxiety. In addition, research has shown that reduced function of the frontal regions of the brain underpins this. This has led to attentional bias modification (ABM) programmes designed to prevent and reduce anxiety. However, other types of negative affect, such as anger, have not received the same attention. This is somewhat perplexing, given that anger is a strong emotion with wide ranging consequences in terms of aggression and this lack of research leaves unanswered questions about whether similar mechanisms underpin aggression. Electroencephalographic (EEG) evidence shows that the hemispheric specialisation of the frontal regions predicts differential motivational responding to emotional stimuli in anger and anxiety. Therefore, when integrated these bodies of literature are subject to theoretical inconsistencies. Importantly, much of the research is correlational so little insight is provided about the causal effects of differential frontal brain asymmetry on attentional bias to threat.

This lack of knowledge raises questions as to whether ABM programmes would be effective in reducing anger and aggression. Manipulating motivational direction, via unilateral hand contractions (UHCs) provides a means to explore the causal effect of increased recruitment of

approach motivated/control networks on attentional bias to threat. Previously this method has only been used to change experiential and expressional aspects of emotion and its effectiveness in modulating attentional components of emotion regulation are unknown.

Therefore, the aims of this Thesis are four-fold. First, it aims to explore whether UHCs would effectively modulate attentional bias to threat. This information is important for establishing whether the UHC method can provide a tool to modulate attentional bias in future research. Second, it aims to establish the effect of the UHC on attentional bias to threat in relation to and in isolation of trait anger. An understanding of this would provide insight into how effective ABM programmes may be in reducing anger. Third, it aims to discover the underlying neural effects of the UHC method in order to examine whether changes in attentional bias to threat reflect modulations in cognitive control and/or approach motivational processes. Finally, a broader aim of this Thesis is to bridge the gap between two large bodies of research in an attempt to provide greater theoretical consistency.

This Chapter firstly presents the previous body of literature on attentional bias in relation to negative affect such as anxiety and anger. The role that frontal brain asymmetry plays in the processing of emotion related stimuli will then be evaluated. The evidence from the attentional bias and frontal brain asymmetry literature will then be converged to highlight gaps in the understanding of how frontal brain asymmetry influences attentional bias to threat. Understanding this relationship is important in working towards resolving theoretical debates between the affective and cognitive literature.

1.1. The Attentional Processing System

Selective attention is typically divided into two systems, an automatic stimulus driven system (e.g. Öhman, 1996; 2005; Öhman and Mineka, 2001; Moors and de Houwer, 2006) and a strategic goal directed system (Shiffrin and Schneider, 1977; Posner, 1980; Posner, Snyder, and Davidson, 1980; Corbetta and Shulman, 2002). The goal directed system has a limited capacity, and processes are effortful, slow, and dependent on conscious awareness and control (Shiffrin and Schneider, 1977; Posner, Snyder, and Davidson, 1980). It allows task relevant information to be selected amidst a myriad of irrelevant distracters in the environment. In an evolutionary sense, this system is crucial for promoting adaptive goal directed behaviours such as finding food or a mate. In order to effectively carry out goal directed behaviour and safeguard this limited capacity system from overload, information irrelevant to the current goal must be filtered (e.g. Lavie, 1995; Lavie, Hirst, Fockert and Viding, 2004).

In contrast, the automatic stimuli driven system allows rapid shifts in attention from a current goal to detect imminent threats in the environment such as negative facial expressions (Öhman, 1996; 2005; Öhman and Mineka, 2001). Compared to the goal driven system, the automatic system is capacity free, effortless, and not dependent on conscious control or awareness (Shiffin and Schneider, 1977; Öhman, 1996; 2005; Öhman and Mineka, 2001). When a threat is detected it receives preferential processing over any on-going goal relevant attention (Hanoch and Vitouch, 2004; Meinhardt and Pekrun, 2003).While research typically divides selective attention into goal directed (e.g. Corbetta and Shulman, 2002) and stimuli driven (e.g.Moors and de Houwer, 2006; Öhman and Mineka, 2001) it is important to note that the boundaries between these systems are described as ‘blurry at best’ as these systems

frequently interact with one another during the processing of emotion (Cisler and Koster, 2010, p211).

1.2. Attentional Bias to Threat

Possibly the most prominent feature of emotion is that the same emotion inducing event, such as a threat in the environment, can produce extremely different patterns of reactivity in different individuals (Siemer, Mauss, and Gross, 2007). For example, in the presence of threat, a complex decision process occurs; either behaviour that promotes appetitive goal attainment is continued or the individual stops the current behaviour and attends to the threat. This requires a complex interplay between cognition and emotion. Therefore, individual differences in emotion based responding is crucial for motivated behaviours and successful adaptation. The next section of this chapter highlights that a preferential attentional processing for threat over neutral stimuli, known as attentional bias to threat, is a prominent feature of negative affect personality characteristics. Furthermore, it discusses how attentional bias to threat impedes the ability to successfully regulate emotions, which subsequently perpetuates attentional bias to threat. It will also illustrate that attentional bias towards threat reduces the ability to successfully carry out goal orientated performance in the presence of threat. This stresses that cognition and emotion are mutually dependent elements of attentional bias to threat and share a bi-directional relationship.

1.2.1. The Role of Attention in Negative Affect

As discussed above normative facilitated threat detection allows rapid responding to high threat in the environment (e.g. the sound of a gunshot) which inhibits on-going tasks to attend and respond to threat efficiently. However, a heightened sensitivity of the automatic

attentional system, resulting in even moderately threatening stimuli receiving preferential processing over neutral stimuli, is known to play a fundamental role in causation and maintenance of negative effect disorders such as anxiety (Beck, 1976; Eysenck, and Calvo, 1992; Eysenck, Derakshan, Santos, and Calvo, 2007; Mathews and MacLeod, 2002; Williams, Mathews and Mackintosh, 1998; Mogg and Bradley, 1998). Indeed, a large body of literature have shown that, compared to healthy individuals, attentional bias towards threat is a prominent feature in individuals with negative affect disorders such as anxiety (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, and van IJzendoorn, 2007; Mogg and Bradley, 1998; Williams, Mathews, and MacLeod, 1996; Mogg and Bradley, 2005). However, before discussing the empirical literature on attentional bias to threat, it is important to first conceptualise the differential processes involved in attentional bias toward threat and theoretical explanations about the influence of individual differences.

1.2.2. Conceptualising Attentional Bias

Preferential processing of threat can be best conceptualised by exploring three inter-related features of attentional bias; the processing stage at which attentional bias occurs, the observed components of attentional bias and the underlying mechanisms of attentional bias. As discussed previously, the attentional system can be divided into the automatic (effortless, capacity free subconscious processing stage) and goal directed systems (effortful, limited capacity conscious stage) (Shiffrin and Schneider, 1977) and it has been demonstrated that attentional bias can be observed in both systems (Cisler and Koster, 2010; Cisler, Bacon and Williams, 2009; Bar-Haim, et al,. 2007; Koster, Crombez, Vershuere, Van Damme, and Wiersema, 2006; Koster, Verschuere, Crombez, and Van Damme, 2005). Further important aspects of attentional bias to threat are its componential characteristics and underlying mechanisms. Visual attention involves three distinct operations, an initial orientating in

attention to the threat stimuli, engaging with the threat stimuli and disengaging attention from the stimuli (e.g., Posner, 1980; Posner and Peterson, 1990). Attentional bias can be observed in all three of these components.

First, attentional bias can be observed at the attentional orientation stage, where threat stimuli are detected faster and with greater ease than neutral stimuli (e.g., Fox, Russo, Bowles, and Dutton, 2001; Öhman et al. 2001; Öhman, 1996; 2005). This is known as vigilance or facilitated attention towards threat. Vigilance towards threat is an automatic process that is suggested to be modulated by the amygdala (Cisler and Koster, 2010). Evidence for the role of this deep brain structure in early vigilance for threat has emerged from studies showing positive associations between amygdala activity and responses to fear (e.g. Phelps and LeDoux 2005; Davis, 2006; Davis and Whalen, 2001; LeDoux, 1996; 2000). Furthermore, it has been suggested that amygdala activation and interconnected fear circuits facilitate the rapid detection of threat for rapid responding (Ohman, 2005) and that anxious individuals show increased amygdala activity during the presence of threat (Stein, Goldin, Sareen, Zorrilla, and Brown, 2002; Stein, Simmons, Feinstein, and Paulus, 2007; Phan, Fitzgerald, Nathan, Tancer, 2006).

Second, attentional bias can also occur at the engaging and switching stages of attention, with threat reducing the ability to disengage attention away from the threat (e.g., Derryberry and Reed, 2002; Koster, Crombez, Verschuere, and De Houwer, 2004; Amir, Elias, Klumpp, and Przeworski, 2003; Fox, Russo, Bowles, and Dutton 2001). Neurocognitive models suggest the inability to disengage attention is associated with impaired top-down attentional control mechanisms that function to inhibit the processing and subsequent responding to threat

(Posner and Rothbart, 2000; Derryberry and Reed, 2002; Eysenck, et al., 2007; Bishop, 2007; 2009; Matthews and Wells, 2000). Furthermore, this impairment is suggested to maintain attentional bias and anxiety (Eysenck and Derakshan, 2011). The underlying neural mechanisms of later disengagement difficulties are suggested to be associated with decreased activation of the prefrontal cortex (PFC), particularly the dorsal lateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC) and the anterior cingulate cortex (ACC) (Bishop et al., 2004; Bishop 2007; 2008; 2009). Evidence has shown that these regions regulate the activity of subcortical structures such as the amygdala and are implicated in the attainment of goal orientated behaviours (Miller and Cohen, 2001). Furthermore, it is widely accepted that the DLPFC functions to control emotion based responding (Miller and Cohen, 2001; Wallis and Miller, 2003; Davidson, 2004). Greater information about the functions of these regions is presented in later sections of this chapter.

Finally, a third source of attentional bias can occur during switching and engagement operations involving avoidance, whereby attention is preferentially allocated away from threat (Koster, et al, 2005; Koster, et al., 2006; Mogg, Bradley, Miles, and Dixon, 2004). Avoidance is particularly important for the goal directed regulation of emotion (Koster, et al., 2006) and functions to reduce, maintain, or increase, negative and positive aspects of experiential and/or expressional forms of emotion (Gross, 1998; 2001, 2002). According to the Process Model of emotion regulation (e.g. Gross, 1998) attentional deployment allows attention to be either purposively allocated towards information that requires elaborated processing or away from unpleasant stimuli to reduce negative affect. The recruitment of the DLPFC is seen as important for this behaviour as cognitive control over emotion is required for this process to ensure successful goal attainment (Miller and Cohen, 2001).

1.2.3. Theories of Attentional Bias to Threat

Numerous theories have been put forward to explain how automatic and goal driven attentional systems interact with one another and how individual differences in emotional experience influence the attention processing of threat (e.g. Bower, 1981; Wells and Mathew, 1994; Beck and Clarke, 1997; Mathews and Mackintosh, 1998). For the sake of brevity, only the models most pertinent to the studies presented in this Thesis are evaluated below to provide insight into how attentional bias to threat influences the regulation of emotion and explain the underlying processes behind attentional bias to threat.

1.2.3.1. The Parallel Distributed Processing Model (PDP; Williams, et al., 1988, 1997)

According to Williams, Watts, Macleod, and Mathews (1988, 1997) all incoming information is assessed and receives a subjective threat value by a pre-attentive affective decision mechanism (ADM). The ADM is assumed to involve the activation of threat-tagged units that represent previous threat experiences and computes the threat value of incoming information, as high or low, by weighting incoming information against previous tagged threat. High levels of negative affect moderate attentional bias to threat, by acting as a catalyst which increases the likelihood that information will be 'tagged' as a threat. When information has been tagged as threat it activates a task demand unit which ensures that the threat receives priority processing. If the incoming information is appraised as low threat, previous resource allocation will be maintained without interference.

According to the PDP model, high negative affect will increase the likelihood that more cognitive resources will be directed towards the threat. In contrast, low negative affect will

direct attention away from the threat. The model also assumes that when people are in a low stress situation (i.e. when state anxiety is low), attentional differences between people with high and low trait anxiety may not be noticeable. However, in high stress situations (i.e. when state anxiety is high), differences in resource allocation between high and low trait anxiety become more apparent. From this perspective, clinical anxiety is determined by the interplay between predisposed personality characteristics (trait anxiety) and situational factors (state anxiety) which mediates attention to threat (Cisler and Koster 2010). This model provides a framework to explain mechanisms that underlie pre-attentive, attentional, and post-attentive threat processing. It also explains the influence that state and trait emotions play in attentional bias. However, the model is suggested to rely heavily on an interaction hypothesis which posits that low trait anxiety individuals avoid threat in any situation (Mogg, and Bradley 1998). However, this is highly questionable given the adaptive functions of the attentional system in threat detection; avoidance in all situations would reduce the ability to rapidly respond to imminent danger (Cisler and Koster 2010).

1.2.3.2. Feature Detection Model (Öhman, 1996; 2005)

According to Öhman (1996; 2005), the automatic detection of potential threat is evolutionary prewired in all human to promote defensive escape (flight) or attack (fight) behaviours following fear. The model assumes that the amygdala plays a central role in automatic, pre-attentive detection of threat and that anxiety modulates its output. Öhman (1996; 2005) suggests that the facilitated detection of threat involves an unconscious, adaptive two-route process, a fast pre-attentive autonomic route and a slower, conscious, significance evaluation route. The pre-attentive route involves the activation of a feature detection system triggered by biologically relevant threat stimuli in the environment (e.g. spiders and snakes). Once activated, the feature detection system then triggers the activation of the autonomic arousal

system. Alternatively, following the activation of the feature detection system, information takes a rapid, automatic, pre-attentive route to the significance evaluation system and then proceeds serially onto the slower, conscious perception system. The conscious perception system functions for the slow appraisal of the available information, achieved through the interaction of the expectancy system that stores emotional memories and prior learning. If information, following the significance evaluation route, is appraised as threat, it then influences the autonomic arousal system, through the means of a feedback mechanism, with increased arousal priming further significance evaluation and the detection of further expected threats. In turn, threat-related stimuli receive increased processing priority, which then leads to attentional bias for threat. While this model provides a clear explanation for vigilance towards threat it, has been suggested that it does not provide a suitable explanation of the processes that underpin attentional capture (Mathews and Mackintosh, 1998).

1.2.3.3. Cognitive-Motivational Model (Mogg and Bradley, 1998)

According to Mogg and Bradley (1998) two functional systems are responsible for attention bias to threat: the valence evaluation system (VES) and the goal engagement system (GES). The VES appraises the valence of incoming information based on its nature, context, and prior learning. The VES output passes forward onto the motivationally driven GES which allocates attentional resources. If information is appraised as threatening by the VES, the GES interrupts the pursuit of current goals and allocates attentional resources to the threat. If information is appraised as non-threatening, any further processing will be inhibited by the GES. It is thought that the motivationally driven GES is evolutionary adapted to detect threat and automatically elicit cognitive, behavioural and physiological responses to interrupt on-going goals and prioritise action to deal with the threat. High trait anxiety is assumed to increase the likelihood that even mild threat information will be tagged as highly threatening

which in turn interrupts the pursuit of goal relevant behaviour. In contrast, people with low trait anxiety will appraise mild threat information as irrelevant and subsequently inhibit further processing of that information. As such, this model assumes that individual differences in attentional bias to threat will only be evident for mild but not high threat information. A major strength of this model is that it highlights the conceptual distinction between the appraisal and goal orientated responses. The model goes further by implicating the amygdala, associated thalamic, hippocampal and cortical structures in appraising the threat stimuli. However, others have suggested that there has been little evidence to support the assumptions that high anxiety is associated with a highly sensitive VES and more importantly the model does not provide an explanation for disengagement difficulties (Cisler and Koster 2010).

1.2.3.4. Attentional Control Theory (Eysenck and Derakshan, 2011)

According to the Attentional Control Theory (ACT) anxiety impairs the processing efficiency of two components of the central executive, namely inhibition and shifting functions. The inhibition function uses negative attentional control to inhibit interference from task irrelevant information or responses. In contrast, the shifting function uses positive attentional control to shift attention between and within tasks. These functions ensure that attention remains focused on task relevant information to maximise performance. It is assumed that deficient attentional control disrupts the balance between stimuli driven and goal driven attention. Specifically, anxiety increases attention to task irrelevant information (threat) and reduces attention to task relevant goals. The ACT also assumes that people with high anxiety use greater strategic effort and processing resources to achieve reasonable task performance (performance effectiveness). The ratio between performance effectiveness and the use of effort/processing resources, to reduce the influence of stimuli driven attention is referred to as

processing efficiency. According to ACT anxiety is characterised by a low processing efficiency and that motivation plays a fundamental role in this. For example, if a task is undemanding or the goal is not clear then motivation is low and people with high anxiety will make minimal use of attention control strategies. Conversely, if the task is demanding and the goals are clear their motivation will be high and greater attempts will be made to override influences of threat information through substantial but inefficient effortful processing and attentional control. A major strength of this model is that it provides an explanation for attentional bias based on attentional control. For example, according to Eysenck et al., (2007) attentional control underpins vigilance for threat by enhancing control over the stimulus driven attentional network and underpins disengagement difficulties by impairing control over the goal directed attentional network.

1.2.3.5. The Integrative Model (Bar-Haim et al., 2007)

Bar-Haim et al., (2007) incorporates several aspects of previous models (Williams et al., 1988, 1997; Mogg and Bradley, 1998) and proposed a multi-dimensional model of threat-related attentional bias. According to this model, incoming information is evaluated by a pre-attentive threat evaluation system (PTES). Information tagged with a high threat value is then passed forward to the resource allocation system (RAS) which elicits physiological arousal, interferes with current task processing, and increases allocation of cognitive resources to the threat. Threat information is then fed forward to a guided threat evaluation system (GTES). This system functions to initiate a strategic assessment of potential coping strategies to deal with the threat, based on previous learning and memory. If information is evaluated as high threat then attention will be maintained on the threat and current pursuit of goals will be interrupted in the goal engagement system (GES). This also increases state negative affect and the primary goal of the individual at this point will be to reduce these feelings. If the

GTSE output is regarded as low threat, this then initiates a feedback mechanism that overrides input originating from the PTES. This reduces physiological arousal, interference and allocation of cognitive recourses to the stimuli and correspondingly reduces state negative affect. According to Bar-Haim et al., (2007) high levels of trait negative affect will result in biases at any of the four stages and that both automatic and strategic levels of information processing contribute to attentional bias. They suggest the automatic threat detection is linked to facilitated attention and that the guided threat evaluation system, which functions to redirect attention to goal relevant tasks, is linked to disengagement difficulties. A major strength of this model is that it provides a clear explanation for vigilance, avoidance, and disengagement difficulties associated with threat.

1.2.4. Attentional Bias Paradigms

Two of the most frequently used paradigms to explore attentional bias to threat in relation to negative affect are the Emotional Stroop Task (EST) and the Dot Probe Task (DPT). In the following section, these paradigms are described in relation to methodological considerations and theoretical implications and findings from studies employing these paradigms are discussed.

1.2.4.1. The Emotional Stroop Paradigm

The most widely used paradigm for capturing and measuring attentional bias in relation to negative affect is the modified EST (Williams et al. 1996). During this task, participants are shown a series of negative (e.g. ‘insult’) and neutral words (e.g. ‘circle’) presented in one of four colours, e.g. red, blue, green and white. Participants are required to indicate the font colour of each word as quickly and as accurately as possible while ignoring its semantic

content. The EST is based on the assumption that preferential processing of the semantic content of threat words (compared to neutral words) will interfere with competing cognitive processing involved in colour responding (Williams et al. 1996; Mathews and MacLeod 1994). This pattern of responding, referred to as the Stroop interference effect, is indexed by slower responses to threat than neutral words.

There are two common variations of the EST: the masked and the unmasked version (e.g. Mogg, Bradley, Williams, and Mathews 1993). The masked EST involves the brief presentation of stimuli (< 20ms) and is assumed to involve preconscious processing of the stimuli. Interference effects found in this version are suggested to reflect automaticity and indicate that attentional bias occurred before conscious recognition. In contrast, the unmasked version of the EST involves the words remaining visible until a response is made. Effects are assumed to reflect later goal directed attentional bias during conscious processing.

Numerous studies have demonstrated that threat words will interfere with task related responses in anxious populations (see Bar-Haim et al. 2007; Mogg and Bradley 1998; Williams et al. 1996). Furthermore, studies exploring the processing stages of attentional bias (utilising masked and unmasked stimuli) have revealed that attentional biases occur at both automatic and strategic processing stages (e.g. van Honk, Tuiten, van den Hout, Putman, de Haan, and Stam 2001; Bradley, Mogg, Millar, and White 1995; Mogg et al. 1993). As such it remains unclear whether attentional bias in anxiety is reflected in early automatic or later cognitive processing stages. Furthermore, while EST provides insight into whether threat-related attentional bias interferes with competing cognitive processes, it cannot provide insight into whether attentional bias reflects facilitated attention or difficulty disengaging (see

Fox 2004 and MacLeod, Mathews, and Tata 1986). For example, the interference effect can be a consequence of both enhanced attention and overall delayed responding to threat (Algom, Chajut, and Lev 2004). It has also been suggested that interference might reflect strategic avoidance of processing threat rather than attentional capture (DeRuiter and Brosschot 1994). Therefore, the EST has received wide criticism (e.g. MacLeod et al. 1986) as it cannot differentiate the componential characteristics of attentional bias i.e. facilitated attention towards threat, difficulty disengaging from threat and attention avoidance of threat.

1.2.4.2. The Dot-Probe Paradigm

The interpretation issues levied at the EST can be overcome using the DPT (MacLeod et al. 1986). The DPT methodology is described in greater detail in Chapter 2 but in brief, in the DPT word-pairs (e.g. threat-neutral) are presented and then immediately after word-pair offset a probe either replaces the threat or the neutral stimuli. Participants are required to respond to the probe location as quickly as possible. It is assumed that responses will be faster when a probe replaces stimuli where attention was allocated prior to probe presentation. Therefore, responses to probes in threat locations are interpreted as vigilance or difficulty disengaging from threat and slower responses to probes in threat locations are interpreted as avoidance (MacLeod et al. 1986).

Research employing this paradigm has typically found that individuals with high anxiety show an attentional bias towards threat compared to neutral stimuli (Mogg, Bradley, De Bono, and Painter 1997; Bradley, Mogg, Falla, and Hamilton 1998; Bradley, Mogg, White, Groom, and de Bono 1999; Pishyar, Harris, and Menzies 2004). Subsequently, research that uses a modified methodology to disentangle the effects of vigilance and difficulty in

disengagement (Koster et al. 2004) have repeatedly shown that attentional bias towards threat reflects difficulty disengaging rather than vigilance for threat (Fox et al. 2004; Koster et al. 2006; Koster, et al. 2004). These findings support the view that cognitive control impairments are an underlying cause of attentional bias in anxiety (e.g. Eysenck and Derakshan, 2011).

There are inconsistencies in the anxiety research employing EST and DPT paradigms. Attentional bias to threat has been observed at both early and later processing stages and has been shown to depend upon the stimuli presentation time and the paradigms employed (See Bar-Haim et al. 2007; Cisler and Koster 2010; Cisler et al. 2009). While it is widely accepted that the EST and DPT both provide a measure of attentional bias to threat (e.g. Driver 2001) it is also generally accepted that they operationalise different characteristics of attention (e.g. Egloff and Hock 2003; Brosschot, de Ruiter, and Kindt 1999; Mogg, Bradley, Dixon, Fisher, Twelftree, and McWilliams 2000). For example, the EST measures responses to simultaneously presented threat and target information while the DPT measure attention to targets presented after the threat is processed. For this reason it has been suggested that the DPT measures attention allocation at a later processing phase than the Stroop task (Brosschot et al. 1999). This may explain differential findings at both early and later processing stages.

The research presented above has provided an important understanding of how attentional bias towards threat influences task relevant performance in individuals with high anxiety. However, a major limitation of the behavioural EST and DPT findings is that they do not provide insight into the underlying neural mechanism of threat-related attentional bias in anxiety.

1.2.5. ERP Indices of Attentional Bias to Threat

In contrast to behavioural reaction time measures, event-related brain potentials (ERPs) provide fine-grained multiple, millisecond measurements of brain activity that can be time-locked to a specific emotional or response related process. Furthermore, exploring the amplitude of individual ERP components provides insight into the extent to which a specific cognitive processing operation is being engaged by a specific task or stimuli (detailed information about the ERP method and ERP components is provided in Chapter 2). Therefore, ERPs provide a more sensitive index to explore the underlying mechanisms of the threat-related attentional bias than RT measures. This assumption is supported by numerous studies that have observed significant differences in ERP measures of attentional bias to threat but no behavioural effects (e.g. Bar-Haim, Lamy, and Glickman 2005; Carretie, Martin-Loeches, Hinojosa and Mercado 2001; Thomas, Johnstone and Gonsalvez 2007; Perez-Edgar and Fox 2003). Exploring individual ERP components provides insight into differential underlying sensory and cognitive processes of emotion-cognitive related responding.

Early attentional processes are reflected in ERP components known as P1 and N1. These represent early rapid automatic orienting of attention (Luck, Heinze, Mangun, and Hillyard 1990; Luck 2005; Hillyard, Mangun, Woldor, and Luck 1995; Mangun 1995; Clark and Hillyard 1996; Hillyard and Anllo-Vento 1998; Fichtenholtz, Hopfinger, Graham, Detwiler, and LaBar 2007). In contrast, more elaborative attentional processing related to evaluation, motivation and inhibition can be indexed by later components namely the P2, N2, P3 and negative slow wave (NSW). For example, the P2 component denotes early global evaluation of emotional and task relevant stimuli that guides behaviour (Schapkin, Gusev and Kuhl, 2000; Kenemans, Kok, and Smulders, 1993; Potts and Tucker, 2001) while the frontocentral

N2b is thought to denote response inhibition and/or conflict monitoring (e.g. Braver, Barch, Gray, Molfese, and Snyder 2001; Jones, Cho, Nystrom, Cohen, and Braver 2002; Nieuwenhuis, Yeung, Van Den Wildenberg, and Ridderinkhof 2003; van Veen and Carter 2002; Dennis and Chen 2007, 2009; Mangun and Hillyard 1995). Later in the attentional process, the P3b component denotes allocation of attentional resources towards motivationally relevant goal target or emotional stimuli (Polich 2007; MacNamara, Foti, and, Hajcak 2009; Duncan-Johnson and Donchin 1977; Johnston, Miller, and Burleson 1986; Nieuwenhuis, Aston-Jones, and Cohen 2005; Duncan, et al. 2009; Johnson 1986). In contrast, the NSW represents conceptual level inhibition and word processing efficiency during EST conflict and interference (Liotti, Woldorff, Perez, and Mayberg 2000; West 2003; West and Alain 2000; Markela-Lerenc, Ille, Kaiser, Fiedler, Mundt, and Weisbord 2004; van Hooff, Dietz, Sharma, and Bowman 2008). For a more detailed discussion about these components see Chapter 2.

Only four EST studies to date have explored ERP indices of attentional bias to threat in relation to anxiety (Thomas et al. 2007; Li, Zinbarg, and Paller 2007; Van Hoff et al. 2008; Taake, Jaspers-Fayer, and Liotti 2009). These studies have shown that attentional bias to threat compared to neutral words was distinguished by enhanced P1 (Li et al. 2007; Taake et al 2009), P2 (Thomas et al. 2007), and P3 (Thomas et al. 2007; Taake et al. 2009) in high compared to low anxiety participants. No effects have been observed for threat compared to neutral words in relation to NSW modulation. However, larger NSW for threat compared to positive words have been observed in high compared to low anxiety participants (Taake et al. 2009). These findings suggest that both early sensory and later motivation related information processing of threat is influenced by anxiety. These findings are in line with models that suggest anxiety is related to vigilance for threat compared to neutral stimuli (e.g. Williams et

al. 1996). However, a lack of NSW amplitude difference for threat compared to neutral words, suggests that attentional bias to threat in anxiety is not related to later inhibition processes as would be proposed by attentional control theories (e.g. Eysenck and Derakshan 2011). Though, as stated previously the EST does not allow for the isolation and exploration of later disengagement components.

To address interpretational issues relating to componential characteristics of attentional bias, a small number of studies have recorded ERPs during the DPT to explore the underlying mechanisms of attentional bias in anxious compared to non-anxious participants (Eldar, Yankelevitch, Lamy, and Bar-Haim 2010; Fox, Derakshan, and Shoker 2008; Helfinstein, White, Bar-Haim, and Fox 2008; Mueller et al. 2009). The DPT evidence has shown that attentional bias to threat compared to neutral stimuli were distinguished by enhanced N1 (Helfinstein et al. 2008), P1 (Helfinstein et al. 2008; Mueller et al. 2009), N2 (Fox et al. 2008), and P2 (Eldar at al. 2010) in high compared to low anxiety participants. These findings indicate that attentional bias to threat in anxiety is characterised by increased early automatic vigilance towards threat and greater recruitment of attentional control as well as increased threat evaluation. The findings also suggest that anxiety related attentional bias is not related to later motivational and cognitive processing.

However, contradictory evidence from research exploring the plasticity of attention-related ERP components after attentional bias modification have found that reduced attentional bias is related to later elaborate processing changes (Eldar and Bar-Haim 2010; O'Toole and Dennis 2012). For example, following training to reduce attentional bias and increase threat avoidance, anxious participants showed reduced P2 and P3 amplitudes and an N2 amplitude enhancement during the DPT (Eldar and Bar-haim, 2010). More recently, O'Toole and

Dennis (2012) explored the effect of attention modification in healthy non-anxious participants who were either trained to attend to threat or avoid threat in the DPT. Participants who were trained to avoid threat only showed reduced P1 to threat. These results suggest that in non-anxious individuals training techniques to increase threat avoidance reduced early automatic vigilance to threat, but not later processes. The divergent findings to those observed by Eldar and Bar-Haim (2010) indicate that individuals with high anxiety may need to recruit more cognitive resources to modify attentional bias than healthy individuals.

1.2.6. Implications of Attentional Bias Research

The evidence presented thus far has indicated that understanding the underlying mechanism of attentional bias to threat in relation to anxiety has important theoretical and practical implications. It has been shown to be reflected in both early and late attentional processing stages (Bar-Haim et al. 2007; Cisler and Koster 2010; Cisler et al. 2009). However, the ERP literature has highlighted differential attentional bias is reflected in early information processing stages (Eldar et al. 2010; Fox et al. 2008; Helfinstein et al. 2008; Mueller et al. 2009) and this may reflect reduced recruitment of motivational and cognitive control mechanisms (Eldar and Bar-haim 2010; O'Toole and Dennis 2012).

Furthermore, this extensive body of anxiety research has led to the emergence of specific theories that posit that attentional bias to threat is implicated in the aetiology and maintenance of anxiety. Moreover, this has in turn led to the emergence of ABM procedures that have been found to effectively reduce anxiety (Heeren, Mogoase, McNally, Schmitz, and Philippot 2015; Amir, Beard, Burns, and Bomyea 2009; Amir, Beard, Taylor, Klumpp, Elias, and Burns 2009; Schmidt, Richey, Buckner, and Timpano 2009; Heeren, Lievens, and Philippot 2011; Heeren, Reese, McNally, and Philippot 2012; MacLeod and Mathews 2012). ABM

aims to reduce the attentional bias towards threat to prevent and treat the symptoms of anxiety (MacLeod and Mathews 2012). Evidence has shown that increased attentional bias to threat is related to the modulation of activity in the DLPFC (Browning, Holmes, Murphy, Goodwin, and Harmer 2010; Heeren, De Raedt, Koster, and Philippot 2013) and that high frequency repetitive Transcranial Magnetic Stimulation (rTMS) on the DLPFC influences the extent that attentional bias is observed (e.g. Leyman et al. 2009; De Raedt et al. 2010). These findings provide further support for the Cognitive Control Model of attentional bias (e.g. Eysenck and Derakshan 2011) in that attentional bias reflects deficits of inhibitory mechanisms. The findings also lead to the assumption that increasing activation of the DLPFC will reduce attentional bias to threat, a suggestion that has direct implications for the use of the UHC method.

1.2.7. Similarities between Anger and Anxiety

Given the scientific contribution of the attentional bias research in informing theory and interventions in relation to anxiety, the importance of this field of research in relation to other forms of negative affect such as anger is highlighted. It is important to note that affective abnormalities such as increased violence and aggression are often co-morbid with negative affect disorders, such as anxiety, depression, bipolar II, and post-traumatic stress disorders (APA, 2000; Kring and Bachorowski 1999; Erwin, Heimberg, Schneider, and Liebowitz 2003; Fava and Rosenbaum 1998; Benazzi 2003 Murphy et al. 2004). Based on the assumptions of the Tripartite Model of emotional disorders (Clark and Watson,1991) that more similarities than variations exist across diverging emotional disorders, research shows similar biological and psychosocial diatheses exist between anger and negative affect syndromes such as anxiety (Barlow 2002; Barlow, Allen, and Choate 2004; Gardener and Moore 2008).

More specifically, similar to anxiety, anger reflects increased negative effect, reduced positive affect, heightened autonomic arousal and perceived uncontrollability (Gardener and Moore 2008; Erwin et al. 2003). It is also assumed that only differences in experiential and behavioural outcomes distinguish between these emotions (Barlow 2002; Barlow, et al. 2004; Gardener and Moore 2008). As a result it is now assumed that anger disorders reflect another facet of negative affect syndrome (NAS) (Barlow et al. 2004). It is suggested that NAS patterns related to previous learning experiences of threat and increased physiological arousal result in an attentional bias for threat, whereby individuals scan their environment for early warnings of threat (Barlow 2002). This anger-related vigilance for external threat is referred to as hostile anticipation and is suggested to be analogue to anxiety related vigilance for threat (Gardener and Moore 2008).

Indeed, evidence has shown that attentional bias towards threat is a prominent feature in domestic violence perpetrators who use violence to reduce exposure to narcissistic threat (Mitchell and Gilchrist 2006). Importantly, the occurrence and magnitude of domestic violence has been suggested to intensify as cognitive distortions and sensitivity towards threat causes increasingly trivial stimuli to be perceived as more threatening over time (Mitchell and Gilchrist 2006). These stimuli may include threat-related facial expressions and/or words. On this basis, it can be assumed that attentional biases may be significant markers of aggression-related cognitive processing and therefore would contribute to the origin, maintenance and consequences of high anger and aggression. Nevertheless, anger has received relatively very little attention in terms of the development of specific theories and even less in mental health settings with regards to clinical conceptualisation and therapeutic interventions.

1.3. Anger-related Attentional Bias

The lack of research exploring anger-related attentional bias to threat is perplexing given the variety of adverse consequences that anger has on individuals and society in general. For example, chronic high levels of anger has been shown to predict an increased likelihood of aggressive behaviour (e.g. Bettencourt, Talley, Benjamin, and Valentine 2006) such as road rage (e.g. Abdu, Shinar, and Merian 2012; Deffenbacher, Lynch, Oetting, and Yingling 2001), aggression in the workplace (e.g. Deery, Walsh, and Guest 2011; Hallberg, and Strandmark 2006; Douglas and Martinko 2001), and domestic violence (e.g. Mitchell and Gilchrist 2006; Barbour, Eckhardt, Davison, and Kassinove 1998). High trait anger has also been shown to predict a number of health issues such as cardiovascular disease (Smith, Glazer, Ruiz, and Gallo 2004; Williams, et al, 2000), stroke (Williams, Nieto, Sanford, Couper, and Tyroler 2002), smoking tobacco (e.g., Spielberger, Foreyt, Goodrick, and Reheiser 1995), high alcohol consumption (Litt, Cooney, and Morse 2000), and unhealthy eating habits (e.g. Anton and Miller, 2005). Furthermore, as attention processing of threat information is well known to play an important role in automatic regulation of emotions (Gross 1998; 2007) it is important to gain a better understanding of attentional processes underlying trait anger as this would have practical implications for informing interventions that focus on the anger-related social and health issues described above.

1.3.1. Conceptualising Anger

Before discussing previous research in the field of anger and attentional bias, it is important to begin by conceptualising anger and discussing important distinctions that are made within the literature. Anger is an internal subjective negative emotion that is usually caused by unpleasant events (Harmon-Jones 2004). A major distinction made in the anger literature is between state and trait anger. State anger is defined as an internal emotional state that

involves subjective feelings caused by insult, injustice, or frustration that range in intensity from mild irritation to intense fury and rage (Spielberger 1999). In contrast, trait anger is defined as the stable metric that reflects individual differences in the frequency, duration and intensity of anger, that people experience in general (Deffenbacher 1992; Spielberger 1999).

Another important distinction that is made in the anger literature is the difference between approach and withdrawal motivated expressions of anger (e.g. Berkowitz 1990; Carver and Harmon-Jones 2009; Harmon-Jones 2004; Watson 2009). Anger expression as indexed by the State Trait Anger Expression Inventory, (STAXI-2; Spielberger 1999) is dependent on individual differences in trait anger expression styles. The propensity to direct anger towards people or objects in the form of verbal or motor behaviour is referred to as approach motivated 'anger expression-out' (AX-O; Spielberger 1999). In contrast, an inhibition of the anger expression or the withdrawal from an anger inducing situations is referred to as withdrawal motivated anger-in (Spielberger 1999). More detailed information on the STAXI-2 can be found in Chapter 2. While high trait anger predicts an increased tendency to experience state anger (Deffenbacher 1992) and to engage in outward behavioural expressions of anger (Bettencourt et al., 2006), it does so only in the perceived presence of a threatening stressor (Davidson 1992; Bettencourt et al. 2006; Deffenbacher 1992). With regards to these distinctions, the studies presented in this Thesis, along with the review within this chapter, focus on trait anger as indexed by trait AX-O in an attempt to explore how individual differences in these anger characteristics influence attentional bias to threat. AX-O was chosen as this is related to the outward expression of anger (Spielberger 1999) as well as to greater approach motivation (Carver and Harmon-Jones 2009) and relative left frontal brain activity (Harmon-Jones, 2004). In contrast, AX-I is related to supressed anger (Spielberger 1999) and greater relative right frontal brain activity (Harmon-Jones, 2004).

1.3.2. Theories of Anger-Related Attentional Bias

In addition to the attentional bias theories presented above (see Section 1.2.3.) a number of cognitive theories have also been developed to explain how attention, memory accessibility, interpretation, and effortful control play a prominent role in anger generation (Anderson and Bushman 2002; Berkowitz 1990; Dodge and Crick 1994; Wilkowski and Robinson 2008). These models share the assumption that the individual variability in the cognitive processing of threat is a major cause of anger and anger expression. However, for brevity this review will focus on models that highlight the importance that attentional bias has in the generation of anger and it behavioural expression in the presence of threat.

1.3.2.1. Social Information Processing (SIP) Theory (Crick and Dodge 1994)

One of the most influential models for explaining individual differences in reactivity to threat is the SIP theory (Crick and Dodge 1994; Dodge 1986). This theory assumes that two processing stages underlie individual differences in approach motivated anger expression in the presence of threat. The first stage involves an early preferential attentional allocation to, and encoding of, threat stimuli in the environment. The second stage involves the greater interpretation of the threat context; according to the SIP theory an attentional bias to threat will increase the likelihood that anger and 'anger expression out' will occur.

1.3.2.2. Cognitive Neo-Associative Model (Berkowitz 1990, 1993)

In contrast, Berkowitz (1990, 1993) postulates that bias in higher cognitive processes explains the generation of anger and aggression. The central focus of the Cognitive Neo-Associative Model is that threat causes 'spreading activation' through an associative network of thoughts, memories and emotions that are associated with previous threat cues. This then

influences interpretations of the threat, but more importantly this spreading activation engages the attentional allocation of cognitive resources towards the threat. According to this model, although not explicitly stated, individual differences in the associative network suggest that threat will be more likely to capture attentional resources in individuals with high anger and that this would cause greater cognitive interference during competing task performance.

1.3.2.3. General Aggression Model (GAM; Anderson and Bushman 2002)

Similar to the Cognitive Neo-Associative Model (Berkowitz 1990, 1993) the GAM (Anderson, Deuser, and DeNeve 1995; Anderson and Bushman 2002; Anderson and Carnagey 2004) suggests that spreading activation processes are a major contributor to anger and anger expression. Furthermore, the GAM posits that prolonged threat interpretation will capture attention, further increasing the intensity and duration of anger experience. This prolonged focus on threat is known as ruminative attention and will increase the likelihood that attentional bias threat will interfere with competing cognitive processes.

1.3.2.4. Integrative Cognitive Model of Trait Anger (Wilkowski and Robinson 2008)

The Integration Model (Wilkowski and Robinson 2008) merges previous models into one conceptual framework to provide a more comprehensive model of anger and the role of attentional bias. It proposes that high state anger and aggression are a result of biases at three separate stages. Firstly, an early automatic interpretation of ambiguous stimuli occurs, with high trait anger individuals interpreting ambiguous cues as threat. Secondly, information interpreted as threat then automatically captures attention (e.g. Ohman 2005). The extent to which attention is captured then influences anger intensity, with prolonged capture leading to

amplified anger and aggression. According to Wilkowski and Robinson (2008), effortful control can be exerted at either the interpretation, attentional or emotional stages, but they suggest that high trait anger individuals show deficient control mechanisms.

These models are influential in providing a framework to understand the importance of early attentional bias towards threat (vigilance) and later attentional capture (disengagement difficulties) in relation to anger. However, insight into the causal relationship between attentional bias and anger remains unclear from the anger literature alone. While the anxiety theories explain attentional bias in relation to anxiety, research that suggests anger and anxiety share similar processing characteristics (Barlow, Allen, and Choate 2004; Gardener and Moore 2008) lead to the assumption that these theories can be applied to the study of attentional bias in anger.

1.3.3. The Anger-Related Attentional Bias Literature

In the following section, a review of previous research that has explored the effects of high anger on attentional bias to threat will be provided. This literature will highlight that high anger increases attentional bias to threat. According to models of anger (Wilkowski and Robinson 2008; Anderson and Bushman 2002; Crick and Dodge 1994) attentional bias to threat should lead to increased anger. However, a handful of studies have now shown the inverse of this whereby anger predicts attentional bias to threat (Eckhardt and Cohen 1997; Smith and Waterman 2003, 2005; van Honk, Tuiten, van den Hout et al. 2001), suggesting a bidirectional relationship between anger and threat-related attentional bias.

One collection of studies has employed an EST paradigm (Williams et al. 1996). Using the EST paradigm six empirical investigations have shown that high trait anger predicts attentional interference to both threat-related facial expressions (Putman, Hermans, and van Honk 2004; van Honk, Tuiten, de Haan, van den Hout, and Stam 2001) and words (Eckhardt and Cohen 1997; Smith and Waterman 2003, 2005; van Honk, Tuiten, van den Hout, et al. 2001). While the findings provide evidence that similar attentional bias to threat exists in anger as in anxiety, as discussed previously in the chapter, the nature of the EST means that there are interpretational complexities surrounding the differentiation componential characteristics of the EST interference effect (MacLeod, Mathews, and Tata 1986; Koster et al. 2004).

Several studies have utilised spatial measures of attention to explore attentional bias in relation to trait anger via the DPT paradigm (MacLeod et al. 1986). Studies using this paradigm have shown high anger individuals show an attentional bias to threat, indexed by faster responding to threat than neutral probes (Smith and Waterman 2003; Wilkowski, Robinson, and Meier 2006) suggesting that attention was preferentially allocated towards threat than neutral words. While these DPT studies provide additional support for the view that high anger is related to increased attentional bias, methodological and analytical issues prevent insight into whether the attentional bias towards threat reflected vigilance or difficulty disengaging (Koster et al., 2004, see Chapter 2 for a detailed discussion on the methodological issues of not using baseline neutral-neutral trials). Furthermore, the behavioural research presented above also provides no insight into the underlying mechanisms of these attentional processing patterns.

ERPs studies may provide a solution to this problem; however, to date only three EST studies have recorded ERP in response to anger-related attentional bias (Stewart, Silton, Sass, Fisher, Edgar, Heller and Miller 2010; Bertsch, Bohnke, Kruk, and Naumann 2009; Bertsch, Böhnke, Kruk, Richter, and Naumann 2011). These studies highlighted that attentional bias in high anger is confined to later evaluative and cognitive processing stages indexed by the ERP components (P2, N2, P3 and NSW).

In masked EST research, high anger participants have shown enhanced P2 for threat (angry, fearful) compared to happy stimuli (Bertsch, Bohnke, Kruk and Naumann 2009; Bertsch, Böhnke, Kruk, Richter, and Naumann 2011). This suggests that high anger individuals show an increase in early allocation of attentional resources and increased evaluation of threat compared to neutral stimuli. It is suggested that threat captures attention more than neutral stimuli in high anger individuals. However, in unmasked EST studies, trait anger did not predict P2 amplitudes for threat compared to neutral and positive words (Stewart et al. 2010). This led the authors to conclude that threat-related attentional bias in high anger individuals was confined to later elaborative and inhibitory processing stages.

Previous research has shown that in high compared to low anger participants, N2 amplitude is larger for threat than neutral stimuli (Stewart et al. 2010). This suggests that in high anger individuals, threat stimuli are a source of greater conflict and as such they require more inhibitory resources to suppress conflict and monitor actions compared to neutral stimuli. As also discussed previously (and in detail in Chapter 2), the parietal P3b is suggested to be mainly insensitive to sensory processing and is enhanced during the allocation of attentional resources towards motivationally relevant information (MacNamara, Foti, and Hajcak 2009).

This assumption is in line with evidence that P3b is larger for target than non-target stimuli (e.g. Duncan-Johnson and Donchin 1977) and for emotionally relevant compared to neutral stimuli (e.g. Johnston, Miller, and Burleson 1986; Keil et al. 2002; MacNamara et al 2009). Indeed, some previous EST research has shown that the P3b is larger for threat than neutral words in high compared to low anger participants (Stewart et al. 2010). This finding suggests that in high anger individuals, threat is more motivationally relevant and therefore receives priority processing and greater allocation of attentional resource than neutral words.

In contrast, others have shown a reverse pattern with larger P3b for neutral than threat stimuli in high compared to low anger participants (Bertsch et al. 2009). However, in line with evidence that P3b is larger for target than non-target stimuli (e.g. Duncan-Johnson and Donchin, 1977) it is also suggested that smaller P3b for threat compared to neutral targets denotes increased task difficulty (target would be colour naming) in the presence of non-target threat (Duncan-Johnson and Donchin 1977; Hillyard, Hink, Schwent and Picton 1973; MacNamara et al 2009). Collectively these P3b results suggest that threat is motivationally more relevant and receives priority processing over competing colour responding processing in the EST paradigm.

The frontal-frontocentral NSW represents conceptual level inhibition and word processing efficiency during EST conflict and interference (e.g. van Hooff, Dietz, Sharma, Bowman, 2008). Research has shown that high compared to low anger individuals, exhibit larger frontal NSW amplitudes for EST threat than neutral words (Stewart et al 2010). The authors suggested that this observation indicated that high anger individuals have a cognitive deficit

where greater attentional effort is needed to override threat-related interference than low anger individuals.

Collectively, the EST ERP findings provide insight into the underlying mechanisms of the threat-related attentional bias in high compared to low anger participants. However, it is important to note that research has yet to explore the neural indices of attentional bias to threat in anger using the DPT. Nevertheless, the findings indicate that in high anger individuals threat receives priority processing due to increased recruitment of evaluative, motivational and inhibitory processing compared to neutral stimuli. According to attentional theories, this increased multifaceted processing of threat then interferes with processing of the limited capacity, strategic attentional system disrupting on-going goal relevant attention (e.g. Hanoch and Vitouch 2004; Meinhardt and Pekrun 2003).

In line with the anxiety literature, this collection of behavioural and ERP studies have shown that high anger, also predicts attentional bias towards threat-related stimuli. The corresponding findings between the anxiety and anger literature are not surprising given that both anxiety and anger reflect increased negative affect, reduced positive affect, heightened autonomic arousal and perceived uncontrollability (Gardener and Moore 2008). Furthermore, anger and anxiety are suggested to differ only in their experiential and behavioural outcomes (Barlow, Allen, and Choate 2004; Gardener and Moore 2008). However, it is important to note that while the anxiety literature highlights that attentional bias is reflected in both early and later information processing stages, the anger literature suggests that attentional bias is confined to later processing stages. Although, a lack of research utilising ERPs means that such inferences are still debatable. Furthermore, the attentional bias literature provides

important insight of how anger increases attentional bias to threat although there are number of caveats that need to be considered. First, much of the past attentional bias research relies on the exploration of the relationship between self-reported measures of anger and attentional bias to threat. Therefore, the correlational nature of the findings in this body of research provides little insight into the causal relationship between anger and attentional bias to threat. In addition, the attentional literature has highlighted a number of similarities between anger and anxiety with respect to attention processing and suggests that the frontal regions of the brain play an important role in attentional inhibition of threat. However, this view is inconsistent with EEG evidence that indicates that differential patterns of frontal brain activity between anger and anxiety play a fundamental role in emotion regulation (e.g. Davidson 2003). It then follows that attentional processing of threat, a key component of emotion regulation, should also differ between anger and anxiety. Ambiguities about the direction of any relationship or questions of causality can be resolved by exploring how changes in frontal brain activity affect attention bias to threat. In the following sections of this chapter a review of the EEG literature highlights distinct differences in the emotion regulation processes between anxiety and anger.

1.4. Frontal Brain Asymmetry of Emotion

Emotion is regulated in the human brain by a multifaceted neural circuit that includes the prefrontal cortex (PFC), anterior cingulate cortex (ACC), amygdala, hypothalamus, hippocampus, insular cortex, ventral striatum, and other interconnected regions. While the interplay between these circuits is important for emotion and motivational processes, this review will focus solely on the role of the PFC in implementing emotion regulation and motivational responses. The reason for this is twofold. First, the PFC is suggested to be the 'centre' for all facets of emotional and motivational processing (Davidson 2004, p220).

Second, due to the methodological restraints of EEG methods, research discussed hereafter focuses on emotion processing in the PFC rather than processes localised to deeper brain circuitry (See Chapter 2 for a detailed discussion of EEG measures of brain activity). Before discussing the literature that has shown differential emotion regulation patterns exist between individual differences in key personality characteristics, the role of the PFC in emotion regulation will be discussed.

1.4.1. Role of the PFC in Emotion Regulation

In emotion-cognitive processing the PFC is theorised to play a fundamental role in maintaining the representations of current and future goals and guiding behaviours for the attainment of these goals (Miller and Cohen 2001). During ambiguous situations the PFC communicates with other regions of the emotional circuit to promote suitable goal relevant behaviours even when competing and sometimes stronger alternative responses are available. There are some significant subdivisions of the PFC that are critical to comment on with respect to emotional processes. First are the distinctions among the interconnected sub-divisions of the PFC and the second is the distinction between left and right hemispheres of the PFC.

With regards to the first distinction, the functions of the PFC are divided into separate anatomical sub-divisions, namely the OFC, ventromedial (vmPFC), and DLPFC (e.g. Rolls 1999). The orbital sector of the PFC is known to be directly associated with assigning affective value to stimuli and affect-guided decision-making based on rewards (O'Doherty, Kringelbach, Rolls, Hornak, and Andrews 2001; Rolls, Kringelbach, and de Araujo 2003; Rolls, and Grabenhorst 2008). The ventromedial sector, specifically the right ventromedial PFC, has been shown to be important for decision making when inhibition of dominant

responses is required in the face of threat and punishment (Tranel, Bechara, and Denburg 2002). In contrast, the dorsolateral PFC receives emotion based input from the orbital sectors (Rolls 1999) and functions to control subsequent reward based emotion responding (Miller and Cohen 2001; Wallis and Miller 2003; Davidson 2004). In turn, the attentional and cognitive influence of the DLPFC feed back to the orbital sectors to modulate affective value of stimuli (Rolls and Grabenhorst 2008).

Based on the evidence presented above it has been suggested that in tasks that require a response (such as the EST and the DPT) emotion based responding would be most reflected in DLPFC activity. It is also important to note that evidence suggests EEG based measures of brain activity reflect activity of the dorsolateral sector of the PFC and that affective neuroscience EEG based findings may reflect cognitive rather than affective components of the PFC (Davidson 2004; Hornak, Bramham, Rolls, Morris, O'Doherty, Bullock, and Polkey 2003). Nevertheless, it is important to convey that the EEG literature on frontal brain asymmetry reflects activity from only a minutia of the underlying processes of the emotion circuitry. Therefore, it has been suggested that patterns of frontal brain asymmetry only contribute and are not sufficient in isolation of complete circuitry activity to produce responses to emotion-related stimuli (Davidson 2004). With respect to the second distinction, research on the hemispheric specialisation of the PFC in emotional processing provides insight into how individual differences influence the experience and expression of emotion and motivational processes. Research exploring the distinction between left and right hemispheres of the PFC will now be discussed in more detail in the following sections.

1.4.2. Models of Frontal Brain Asymmetry of Emotion

1.4.2.1. The Valence Hypothesis

Research exploring the relationship between frontal brain asymmetry and emotional processing gained momentum following observations that lesions to the left frontal brain resulted in depression (e.g. Gainotti 1972; Robinson and Price, 1982), whereas damage to the right frontal brain led to mania (Sackeim et al. 1982). These observations led to the emergence of the affective-valence model of frontal brain asymmetry (e.g. Davidson, 1984, 1998; Ahern and Schwartz 1985; Gotlib, Ranganath, and Rosenfeld 1998; Heller and Nitschke 1998). This model postulates that the left PFC is specialised for the affective experience of positive emotions such as happiness and surprise while the right PFC is specialised for negative emotions such as sadness, fear, and anger.

A substantial amount of EEG research has provided support for the valence hypothesis, showing that greater relative right frontal brain activity at baseline provides a trait-like index of negative affect (e.g. Tomarken, Davidson, Wheeler, and Doss 1992), as well as depression and anxiety (e.g. Henriques and Davidson 1990; Heller and Nitschke 1998; Blackhart, Minnix, and Kline 2006). EEG research has also shown that baseline frontal EEG activity predicts emotional reactivity to emotional stimuli. For example, greater relative right frontal brain activity has been shown to predict a greater incidence of negative affect during the viewing of a fear inducing film clip and less positive affect to a happiness inducing film clip (Tomarken, Davidson, and Henriques 1990; Wheeler, Davidson, and Tomarken 1993). Greater right frontal brain baseline activity has also been shown to predict crying following maternal separation in infants (Davidson and Fox 1989).

In addition, research employing phasic measures of EEG has shown that frontal brain activation can be modulated by emotion related stimuli. For example, viewing or performing happy compared to sad facial expressions will increase relative left frontal brain activity (Davidson and Fox 1982; Ekman and Davidson 1993). In contrast, increased relative right frontal brain activity is induced following voluntary fearful facial expressions (Coan, Allen, and Harmon-Jones 2001) and taste induced disgusted facial expressions (Fox and Davidson 1986).

Collectively the studies discussed above demonstrate that positive and negative affect at both trait and state levels are associated with increased left and right frontal brain activity respectively. These findings have also been theoretically replicated using ERPs (Cunningham, Espinet, DeYoung, and Zelazo 2005), and functional magnetic resonance imaging (fMRI) (Canli, Desmond, Zhao, Glover, and Gabrieli 1998). However, more recent evidence suggests that the valence hypothesis does not provide a sufficiently accurate explanation of the hemispheric specialisation of emotional processing and that motivational direction might offer a more appropriate explanation.

1.4.2.2. The Motivational Hypothesis

The Motivational Model (Davidson 1998; Davidson and Irwin 1999; Davidson, Marshall, Tomarken, and Henriques 2000; Fox 1991; Harmon-Jones and Allen 1997; Sutton and Davidson 1997) states that the anterior hemispheric specialisation of the brain is based on the experience and expression of emotions that motivates and guides movement towards

(approach) or away (withdrawal) from an emotion related stimulus during goal attainment. According to this theory left anterior regions are implemented in the experience and expression of positive approach-related emotions and the right in the experience and expression of negative withdrawal-related emotions. In line with contemporary theories of emotion, that suggest that positive affect is associated with approach motivation and negative affect is associated with withdrawal motivation (e.g. Gray, 1990; Watson, 2000), the Motivational Model assumes that the approach motivational system promotes appetitive behaviour and positive affect such as happiness and enthusiasm. In turn, this facilitates movement towards appetitive goals. In contrast, the withdrawal motivational system promotes withdrawal behaviours and negative affect such as fear and disgust in order to facilitate movement away from aversive stimuli. Davidson (2004) went further to suggest that left frontal regions are involved in approach motivated, goal directed, action planning, and responding and the right frontal brain is important for inhibiting behaviour and promoting vigilance towards threat.

Research exploring the role of frontal brain asymmetry in motivated responding has typically indexed approach and withdrawal motivation through the use of the behavioural inhibition/behavioural activation system (BIS/BAS) scales (Carver and White, 1994). This scale was based on Gray's (1994) Motivational Theory, which argues that the approach motivated behavioural activation system (BAS), motivates behaviours towards goals that promote reward and non-punishment and escape from punishment. In contrast, the withdrawal related behavioural inhibition system (BIS) is theorised to inhibit on-going behaviour, increases arousal and attention towards threat, in preparation for vital movements during conditioned punishment, non-reward and innate fear. Through the application of this theory, substantial evidence has supported the role of the left frontal brain in state and trait

approach motivation (e.g. Harmon-Jones and Allen 1997; Sutton and Davidson 1997; Amodio, Master, Yee, and Taylor 2008; Coan and Allen 2003; Peterson, Gable, and Harmon-Jones, 2008). However, evidence for links between right frontal brain activity and withdrawal motivated response has been somewhat inconsistent, with some showing evidence of a link between right frontal brain activity and withdrawal motivation (Peterson, Gable, and Harmon-Jones 2008; Sutton and Davidson 1997) and others showing no relationship (Harmon-Jones and Allen 1997; Hewig, Hagemann, Seifert Naumann and Bartussek 2006; Amodio et al. 2008; Coan and Allen 2003).

These studies highlight that frontal brain asymmetry may be related to motivational direction rather than valence. However, as discussed previously, approach (BAS) motivation is mostly associated with positive affect whereas withdrawal (BIS) motivation is associated with negative affect (Carver and White 1994). Therefore, previous research has confounded valence and motivation. For example, evidence supporting the valence hypothesis (e.g. Coan, Allen, and Harmon-Jones 2001; Fox and Davidson 1986; Davidson and Fox 1982; Ekman and Davidson 1993) is consistent with the Motivational Model and its' supporting evidence (e.g. Harmon-Jones and Allen 1997; Sutton and Davidson 1997). As such, interpretations of the results from previous EEG research can be viewed from both the valence and motivational hypothesis perspectives, and conceptual explanations are tangled. Therefore, the evidence presented thus far is unable to shed light on whether frontal brain asymmetry reflects emotional valence, motivational direction, or a combination of the two. The next section illustrates that research exploring anger-related frontal brain asymmetry provides a means to detangle these explanations.

1.4.3. Resolving Theoretical Conflict

While in many instances positive and negative affect are generally associated with approach and withdrawal related motivation (e.g. Gray 1990; Watson 2000), anger does not conform to this relationship. Anger is experienced as a negatively valenced emotion that characteristically induces approach motivated behavioural tendencies (e.g. Carver and Harmon-Jones 2009; Harmon-Jones 2004; Watson 2009; Lazarus 1991; Ekman and Friesen 1975; Plutchik 1980). Evidence suggests that both state and trait anger is positively associated with trait approach motivation, indexed by the BIS/BAS scale (see Carver and Harmon-Jones 2009 or Harmon-Jones et al. 2010, for a review). In addition, evidence has also shown that trait BAS is positively associated with behavioural aggression and that this relationship becomes even stronger when approach motivation is initially primed (Harmon-Jones and Peterson 2008; Peterson, Shackman, and Harmon-Jones 2010).

Given that the evidence presented above has shown that anger is positively associated with approach motivation, research has recently focused on exploring the role of frontal brain asymmetry in relation to anger. The aim of this is to provide insight into whether frontal brain asymmetry reflects valence or motivational direction. For example, if anger is related to greater relative left frontal brain activity then it can be assumed that frontal brain asymmetry reflects motivational direction. In contrast, if anger is related to greater relative right frontal brain asymmetry, it can be assumed that frontal brain asymmetry reflects emotional valence.

Substantial evidence in support of the Motivational Model has shown that trait anger is related to increased left frontal activity at baseline (Harmon-Jones and Allen 1998; Harmon-Jones 2004; Rybak et al. 2006). In order to rule out the possibility that this pattern of activation did not reflect positive attitudes towards anger, research controlled for attitudes

towards anger and showed that the relationship between anger and greater left frontal brain activity did not reflect positive attitudes towards anger (Harmon-Jones, 2004). Whilst such evidence is compelling, it is important to note that it was based on correlational designs that utilised self-report measures of trait anger and EEG baseline measures. Therefore, the findings from the studies presented above provide no insight into the causal direction of the relationship between greater relative left frontal brain asymmetry and anger.

To resolve this weakness others have explored the effects of induced state anger on frontal brain asymmetry (Harmon-Jones and Sigelman 2001; Harmon-Jones, Peterson, and Harris 2009; Harmon-Jones, Vaughn-Scott, Mohr, Sigelman, and Harmon-Jones 2004). For example, both anger inducing insult (Harmon-Jones and Sigelman 2001; Harmon-Jones, et al. 2004) and anger inducing jealousy (Harmon-Jones et al. 2009) have been observed to increase relative left frontal brain activity. Studies highlighting this causal relationship between increased state anger and left frontal brain activity contradict the valence hypothesis but support the Motivational Model.

Additional support for the Motivational Model has emerged from findings that reveal that anger-related left frontal brain activity is only evident when state anger is accompanied by an opportunity to approach the anger inducing stressor (Harmon-Jones, Sigelman, Bohlig, and Harmon-Jones 2003; Harmon-Jones, Lueck, Fearn, and Harmon-Jones 2006). In contrast, other evidence has shown that high, compared to low, trait anger participants exhibit increased left frontal brain activity after viewing anger-inducing stimuli even when an opportunity to approach the stressor was not available (Harmon-Jones 2007). This finding is in line with the view that high anger individuals have more extensive anger-related brain networks and that an anger stressor should activate these networks more readily, regardless of

the opportunity to respond (Berkowitz 1993; Berkowitz and Harmon-Jones 2004). Together, these findings indicate that an approach motivated opportunity simply serves to intensify greater relative left frontal activity in relation to trait anger. In contrast, an approach opportunity is essential to increase left frontal brain activity in relation to state anger.

Overall, this provides strong support for the Motivational Model of frontal brain asymmetry, highlighting the relationship between greater left frontal brain activity and approach motivation. However, a major limitation of this work is that it has provided no insight into whether frontal brain asymmetry makes a causal contribution to approach motivated behaviour. More recently, research has begun to manipulate frontal brain asymmetry to explore whether increasing left frontal brain activity would affect approach motivation and aggression (Harmon-Jones 2006; Peterson, Shackman, and Harmon-Jones 2008). Importantly this research provides some of the first evidence that inducing greater left frontal brain activity, through contractions of the right hand, increases both approach motivation to positive stimuli and increases aggression. The hand contraction method employed in the studies by Harmon-Jones (2006) and Peterson et al. (2008) is central to the studies in this Thesis and therefore will be discussed in greater depth in later sections of this chapter.

The research presented above has highlighted that anger and approach motivation are related to increased left frontal brain activity in response to affective experience and expression. However, as discussed previously it is possible that the EEG findings presented above may also reflect cognitive rather than just affective components of the PFC (Davidson 2004). Cognitive processes such as attention play a fundamental role in the generation of emotional experience and expression (Gross 1998; 2002). However, the research discussed so far provides no insight into the influence of greater left frontal brain activity and cognitive

components of anger and approach motivational tendencies. Taken together, associations between anger, motivation, cognitive control and frontal activity suggests that left PFC may play a role in threat-based bias. Furthermore, the research described above suggests that increased left frontal activity would moderate threat-related attentional bias. However, gaps remain in the understanding of hemispheric specialisation of cognitive responses to affective stimuli, especially with regards to attentional bias to threat.

1.5. Modifying Attentional Bias to Threat

To date only one study has explored the causal effect of increased left frontal brain activity on attentional bias (d'Alfonso, van Honk, Hermans, Postma, and de Haan 2000). This study employed the use of slow repetitive transcranial magnetic stimulation (rTMS) to inhibit activity in the left or right DLPFC. When slow rTMS is applied to the right PFC it inhibits cortical activation of this region, causing the left frontal brain to be more active in comparison. In contrast, when slow rTMS is applied to the left PFC it inhibits cortical activation of this region, causing the right frontal brain to be more active. This rTMS study showed greater left DLPFC activation resulted in the preferential processing and selective attention towards angry faces in an EST paradigm while greater right activity produced avoidance of angry faces. Therefore, this provides further evidence that the increased left frontal brain activity induced approach motivation toward threat-related stimuli while increased right frontal brain activity increased withdrawal related behaviour.

However, this observation challenges the view that greater left frontal activity is associated with increased cognitive control (Davidson 2004). It also challenges the view that BIS related increased right frontal brain activity heightens attention towards threat (Gray 1987; Gray and

McNaughton 1996). According to these views it would be expected that increased left frontal brain activity should instead reduce attentional bias towards threat. Interestingly, previous event-related fMRI research (MacDonald, Cohen, Stenger, and Carter 2000) has shown that greater left dorsolateral prefrontal cortex activity was associated with less EST colour naming conflict. This provides further support that the left PFC is implemented in cognitive control required to maintain task related attention. Indeed, it has been suggested that approach motivated left frontal brain activity found in previous anger research may reflect the increased recruitment of the approach motivational system to aid anger reduction (Miller and Cohen 2001). This would further explain why previous research has shown increased left frontal brain activity was only evident in state anger and was dependent on the opportunity to resolve the anger inducing situation (Harmon-Jones et al. 2003).

While the research by d'Alfonso, et al. (2000) supports the affective literature that shows that anger-related left frontal brain activity is associated with approach motivation (e.g. Harmon-Jones and Allen 1998; Harmon-Jones 2004; Harmon-Jones and Sigelman 2001; Harmon-Jones et al. 2009; Harmon-Jones et al. 2004), the findings are inconsistent with the cognitive literature (Davidson 2004; Miller and Cohen 2001). However, an important limitation of rTMS, is that it does not reliably localise the DLPFC (Herwig, Padberg, Unger, Spitzer, and Schonfeldt-Lecuona 2001). This is due to the behaviourally silent nature of the PFC in producing observable responses during rTMS (Penfield 1958) and interindividual differences in the brains functional architecture or the correspondence between scalp positioning and the underlying brain anatomy (Sack, Kadosh, Schuhmann, et al. 2008; Herwig, Satrapi, and Schonfeldt-Lecuona, 2003). Advances in TMS research has led to the emergence of on-line real time neuronavigation systems that allow specific sites such as the DLPFC to be targeted though the use of individual fMRI data (Sack, et al. 2008). However, d'Alfonso, et al.

(2000) localised the stimulation of the DLPFC based on Tailarach Atlas coordinates and the 10-20 system of electrode positioning as set out by previous research (e.g. Pascual-Leone, Rubio, Pallardo and Catala, 1996). This approach did not take into account interindividual differences discussed above (Sack, et al. 2008; Herwig, et al. 2003). Therefore, the degree to which DLPFC activity was modulated is somewhat questionable. This may explain the discrepancies found between the affective and cognitive literature. Despite these issues, the rTMS study does provide a clear rationale to employ alternative techniques, such as the Unilateral Hand Contraction (UHC) method, to induce differential patterns of PFC activity in order to investigate attentional bias in relation to frontal brain asymmetry of emotional processing. The next section provides a brief overview of the UHC method along with empirical evidence to support its use in modulating attentional components of emotion regulation.

1.6. The Unilateral Hand Contraction (UHC) Method

1.6.1. Overview of UHC Method

The UHC method (Schiff and Lamon 1994) involves sustained squeezing of a ball in the right or left hand, which has been suggested to cause an initial activation increase in the contralateral motor cortex that then spreads to contiguous mid-frontal DLPFC regions (Harmon Jones 2006). Detailed methodological information about the UHC is presented in Chapter 2. A number of studies have shown that the UHC method reliably increases contralateral lateral frontal and frontal–central EEG activity (Harmon-Jones 2006; Peterson, Gravens, and Harmon-Jones 2010; Peterson, Shackman, and Harmon-Jones 2008).

1.6.2. The UHC Method in Emotion Modification

The UHC has been reliably shown to modulate emotional experience and provides support for the role of the frontal brain in affective valence. For example, contractions of the right hand were shown to induce positive affect, confidence and bias perceptions and judgments positively while contractions of the left hand induced sadness and biased perceptions and judgments negatively (Schiff and Lamon 1994; Schiff and Truchon 1993).

More recently, the UHC method has been used to explore the relationship between frontal brain asymmetry and motivation direction. For example, Harmon-Jones (2006) explored the effect of right (RHCs) and left (LHCs) hand contractions on approach motivation towards mildly positive stimuli. Findings revealed that UHCs influenced approach oriented emotions, with RHCs inducing greater self-reported approach emotion to mildly positive stimuli than LHCs. This provided evidence that approach motivational systems of the left frontal brain were primed by RHCs (Harmon-Jones 2006). However, a major limitation of this research was that it confounded valence and motivation and as such the findings were in line with both the Valence and Motivational Models of frontal brain asymmetry. To resolve this issue, Peterson et al. (2008) explored the effect of UHC on aggressive responses following insult. This study found that RHCs significantly increased approach motivated aggression, providing some of the first causal evidence that priming the left frontal brain increased approach motivation.

While the UHC method has been shown to modulate emotional and motivational behaviours (Harmon-Jones 2006; Peterson et al. 2008), it is uncertain which antecedent-focused component of emotion regulation was modulated to influence approach motivation. As discussed previously, emotions can be regulated at situational selection, situational

modification, attentional deployment, and appraisal stages before experiential, behavioural, or physiological responses occur (Gross 1998, 2001, 2002). Given that the situation was controlled in the Peterson et al. study (2008) it can be assumed that the increased approach related behaviour following RHCs may have been a result of modifications in the attentional deployment stage of emotion regulation.

Furthermore, as evidence has consistently shown that attentional bias towards threat is important in the aetiology and maintenance of anger and aggression related emotion disorders, an investigation of the effects of RHCs on attentional bias towards threat is warranted. Additionally, although considered a reliable method for inducing approach motivation, research showing a positive association between increased DLPFC activity and cognitive control (Davidson 2003; Davidson 2004; Miller and Cohen 2001) would predict that RHCs would modulate emotion circuits that control anger-related attentional bias to facilitate goal directed behaviour. However, no research has examined the effects of RHC on the cognitive processing of threat.

.

1.7. Summary

Collectively, the literature presented in this review has highlighted that attentional bias towards threat plays an important role in approach motivated anger. It has also demonstrated that understanding the role of the left frontal brain in approach motivated responding has important practical and theoretical implications. Therefore, understanding the neural mechanisms that underlie attentional bias towards threat is fundamental for understanding how and why humans respond to emotional stimuli in general and for understanding

corresponding anger-related behaviours. To date there are still inconsistencies between the affective and cognitive literature with respect to the role that the left frontal brain plays in attentional bias, anger and approach motivation. Furthermore, a gap in the UHC literature warrants further exploration of the effectiveness of RHCs to modulate attentional components of emotion regulation.

1.8. Aims of Research

The aims of the experimental chapters in this Thesis are to provide a novel insight into the effectiveness of the UHC method in the modulation of attentional bias to emotion-related words and how trait anger influences its effects in a healthy sample. Furthermore, the Thesis also aims to provide information about the causal relationship between the increased activation of anger networks and attentional bias to emotion related words. To date research exploring attentional bias to threat in relation to high trait and state anger has been correlational and therefore has provided little insight into the causal relationship between increased activity of anger-related networks and attentional bias to threat. This information is important for establishing whether the UHC method can provide a tool to modulate attentional bias in future research. This information will also provide significant insight into how effective existing intervention programmes may be in reducing anger.

Furthermore, the studies in this Thesis aim to utilise a number of different methodologies to explore the effect of the UHC method in modulating attentional bias to emotion-related words ranging from EST analysis of attentional bias to threat (Chapters 3 and 4), DPT analysis of attentional bias to emotion related words (Chapters 5 and 6), the analysis of ERP measures of cognitive components of attentional bias to emotion related words (Chapters 4

and 6) and self-reported measures of trait anger (Chapters 4, 5 and 6). This will enable the examination of the effects of the UHC on attentional bias modifications across diverse responses and neural domains. In doing so the Thesis aims to provide novel insight and a comprehensive assessment of the effects of UHC method on attentional bias to emotion related words in relation to and in isolation of trait anger.

In addition, two of the studies in this Thesis aim to explore the effect of the UHC method on attentional bias to high arousing emotion-related words presented with low arousal neutral words (Chapters 5 and 6). This allows for a comprehensive examination of whether UHC modulation of attentional biases reflects valence or arousal and ensures that valence and arousal were not confounded. As such the Thesis also aims to provide novel insight into whether the UHC method modulates arousal or valence dimensions of attentional bias.

Overall, the studies presented in this Thesis provide a novel, but comprehensive account of how the UHC method and the effects of anger modulate valence and arousal dimensions of attentional bias to threat across different attentional bias paradigms. In doing so it bridges the gap between two large bodies of research in an attempt to provide greater theoretical consistency.

CHAPTER 2

GENERAL METHODS

2.1 Introduction

The purpose of this chapter is to provide a comprehensive account of the main methods used in the four studies presented in this Thesis. Information regarding the specifics of each experimental study will be presented in the relevant chapters.

2.2 Participants

2.2.1 Inclusion and Exclusion Criteria

To be eligible for participation in the four studies presented within this Thesis, all volunteers were required to be right-handed, assessed by scoring a laterality quotient (LQ) of 100 in the Edinburgh Handedness Inventory-revised (EHI-r; Williams 1986). Participants were also required to be English speaking and have normal or corrected to normal visual acuity. Volunteers were excluded, assessed using a yes/no response format, from participating if they reported having a history of psychiatric disorders, traumatic brain injury, or central nervous system dysfunctions such as epilepsy.

2.2.2 Recruitment and Ethics

Participants were recruited using an opportunity sample of psychology students from Coventry University, UK. Participants were recruited via the SONA participant recruitment

scheme for the sampling phase of the study. In this phase, a questionnaire pack was sent to participants to gather information relating to the inclusion criteria and demographic details. The pack also contained the State Trait anger Expression Inventory STAXI-2 (Spielberger 1999). Scores from these questionnaires were used to recruit participants for the studies within this Thesis. Coventry University Ethics Committee approved all of the studies within this Thesis: written informed consent was gained from each participant and credits were awarded through the SONA participant recruitment scheme.

2.3 Individual Difference Measures

2.3.1 Right Handedness

Right-handedness was assessed using the revised 8-item Edinburgh Handedness Inventory-*revised* (Williams 1986), a modified version of the Oldfield (1971) Edinburgh Handedness Inventory. This assessed the dominance of an individual's handedness when they are writing, throwing, using scissors, a toothbrush, a knife without a fork, a spoon, a computer mouse or when striking a match. The scoring of the revised EHI ranges from -50 (always left) to +50 (always right). The eight activities are then summed to produce laterality quotient (LQ) of between -400 and +400, which is then divided by 4 to provide an Edinburgh LQ. This score ranges from -100, which reflects complete left-handedness to +100 reflecting complete right-handedness.

2.3.2 Trait Anger Expression-Out

Anger was assessed using the revised 57-item State-Trait Anger Expression Inventory STAXI-2 (Spielberger 1999). The STAXI-2 assesses individual differences in state anger,

trait anger and anger expressive styles. The STAXI-2 has been shown to have good reliability, with alpha coefficients ranging from .81 to .93 (Spielberger 1988) and high validity with good psychometric properties (Deffenbacher 1992; Fuqua, Leonard, Masters, Smith, Campbell and Fischer 1991; Spielberger 1988; Spielberger et al. 1995).

The first section of the STAXI-2 assesses state anger (*S-Ang*), defined as a subjective feeling caused by insult, injustice, or frustration that range in intensity from mild irritation to intense rage (Spielberger 1999). There are 15 items used to quantify three distinct components of *S-Ang*; feeling of anger (*S-Ang/F*), the expression of verbal anger (*S-Ang/V*) and the expression of physical anger (*S-Ang/P*). *S-Ang/F* measures the anger intensity felt by the individual at that moment, *S-Ang/V* measures the intensity to express anger verbally at that moment and *S-Ang/P* measures the intensity to express anger physically at that moment. Statements such as, "right now I feel annoyed" (*S-Ang/F*), "I feel like swearing" (*S-Ang/V*) and "I feel like hitting someone" (*S-Ang/P*) are scored on a four point scale, ranging from almost never (1) to almost always (4). These items assess the intensity of anger felt by the individual at that moment in time and range from *not at all* (1) to *very much so* (4). Scores in the *S-Ang* section are added together and produce scores ranging from 15 (indicates low state anger) to 60 (indicates high state anger).

The second section of the STAXI-2 assesses trait anger (*T-Ang*), defined as the frequency that an individual experiences angry feelings in general (Spielberger 1999). This section is made up of eight items that measure angry temperament; (*T-Ang/T*) and angry reaction (*T-Ang/R*). *T-Ang/T* measures the disposition of an individual to experience anger without an explicit provocation while *T-Ang/R* measures the frequency that anger is experienced by the

individual following criticism and frustration. Statements such as, "in general, I am quick tempered" (*T-Ang/T*) and "I feel like hitting someone when frustrated" (*T-Ang/R*) are scored on a four point scale, ranging from almost never (1) to almost always (4). Scores in the *T-Ang* section are added together and range from 10 to 40, with high scores indicating a greater incidence of trait anger. Subsequently high trait anger is indicative of experiencing more frequent and intense increases in state anger during subjectively perceived anger provoking situations (Spielberger 1999).

The final section of the STAXI-2 assesses the way individuals react during the experience of anger (Anger reaction; *T-Ang/R*). This section is broken down into 4 distinct components; Anger expression out (*AX-O*), anger expression in (*AX-I*), anger control out (*AC-O*), and anger control in (*AC-I*). *AX-O* measures the frequency that the individual expresses angry feelings either verbally or physically while *AX-I* measures the frequency that anger is experienced but not expressed (supressed). In contrast, *AC-O* measures the frequency that the individual controls verbal or physical expressions of anger, while *AC-I* measures the frequency that the individual internally reduces the intensity of angry feelings. For this section, participants respond to how they generally react when angry or furious to statements such as "I say nasty things" (*AX-O*), "I boil inside but don't show it" (*AX-I*), "I try to relax" (*AC-I*) and "I control temper" (*AC-O*). Similar to the other sections, responses are scored on a four-point scale, ranging from almost never (1) to almost always (4). Scores in the *T-Ang/R* section are added together and range between 8 and 32. Scores at the lower end of the scale are interpreted as a low incidence of either, anger out, anger in, control out and control in. In contrast, higher scores reflect a higher incidence of the measured component.

Collectively, the 32 items in the *T-Ang/R* section form an anger expression index (*AX Index*) which is used to assess the individual's total anger expression based on AX-O, AX-I, AC-O and AC-I scales. Items in this section are added together and scores range from 0 to 96, with higher *AX Index* scores reflecting individuals that experience intense anger that is either expressed or supressed, or both.

2.4 The Unilateral Hand Contraction (UHC) Method

In all studies within this Thesis, the UHC method (Schiff and Lamon 1994) was used to modulate attention bias to threat. During this procedure participants received onscreen instructions to relax either their right or left hand, depending on group or condition, for 15s and then squeeze a 5cm diameter rubber ball for 45s, completing one squeeze every second. This procedure was repeated an additional 3 times so that the UHC method lasted 4 minutes (as determined by Schiff and Lamon 1994). Within the studies presented in this Thesis, RHCs refers to contraction of the right hand and LHCs refer to contractions of the left hand. During the control condition of no hand contractions (NHCs), participants received a four minute on-screen instruction that guided them to relax and place both of their hands flat on the table with their palms facing down for four minutes (Schiff and Lamon 1994).

As discussed in Chapter 1, The UHC method (Schiff and Lamon 1994) is suggested to increase contralateral motor cortex activity that migrates to contiguous DLPFC regions (Harmon-Jones 2006). Research has shown that the UHC method reliably increases contralateral frontal and frontal–central electroencephalography activity (inverse of alpha) (Harmon-Jones 2006; Peterson, Gravens, and Harmon-Jones 2010; Peterson, Shackman, and

Harmon-Jones 2008). For example, Harmon-Jones (2006) explored the effects of UHC on the activation of right and left frontal cortices by recording EEG activity while participants contracted their right and left hand. In line with evidence that unilateral muscle contractions were associated with contralateral motor activation (Andrew & Pfurtscheller, 1997; Pineda, 2005), results showed that right compared to left-hand contractions caused greater relative left frontal and central activity. As would be expected, effects were stronger at central than frontal sites. The author interpreted this finding as evidence that UHCs caused mid-frontal activation, via cortico-cortical networks between the motor cortex and the DLPFC. However, source generators could not be established in Harmon-Jones (2006) experiment because too few electrodes were available and the involvement of regions such as the premotor cortex, an area localised just anterior to the primary motor cortex was not considered. For this reason, the causal path of the spreading activation between the motor cortex and the DLPFC, following UHCs, remains unclear and it is important to consider that the direction of activity may be reversed or bidirectional.

Similarly Peterson, et al (2008) explicitly examined the spreading activity from central to frontal sites following UHCs through EEG coherence analysis. This EEG analysis technique provides an index of the linear relationship between two distinct scalp sites within a given frequency band with high coherence occurring for regions connected by white matter tracts (Thatcher, Krause, & Hrybyk, 1986). Results demonstrated that RHCs caused greater coherence between the left motor cortex and left PFC while LHCs caused greater coherence between left motor cortex and left posterior regions. Similar to previous research (Harmon-Jones 2006), the direction of causality could not be established but results provided further evidence that RHCs increase activation of the contralateral PFC.

To date no research has explored the effect of UHC method on attentional bias to emotion related stimuli. However, UHCs have been shown to reliably induce changes in experiential and behavioural emotional response (e.g. Schiff and Lamon 1994; Harmon-Jones 2006; Peterson et al. 2008). As discussed in Chapter 1, LHCs induce sadness and negatively bias perceptions and judgments while RHCs induce positive affect, as well as positively bias perceptions and judgments (Schiff and Lamon 1994). More recently, evidence has shown that RHCs prime approach motivational systems and increase approach motivated responding towards mildly positive stimuli (Harmon-Jones 2006). RHCs have also been found to increase self-reported anger and aggression in healthy participants (Peterson, et al. 2008; Peterson, et al. 2010).

These findings support evidence that greater relative left frontal brain activity is positively associated with positive affect (e.g. Davidson 1984, 1998), approach motivation (e.g. Davidson and Irwin 1999; Harmon-Jones and Allen 1997) and aggression (e.g. Harmon-Jones and Allen 1998; Harmon-Jones 2004). Therefore, it can be assumed that RHCs provide a reliable method for inducing approach motivated attentional changes. To date, findings regarding LHCs have only provided support for the Valence Hypothesis that posits that the right frontal brain is implicated in negative affect (e.g. Davidson 1984). While the right frontal brain is associated with withdrawal motivation (e.g. Sutton and Davidson, 1997), albeit somewhat inconsistently (e.g. Harmon-Jones and Allen 1997; Hewig et al. 2006), research has yet to explore whether LHCs are related to motivational direction. Therefore, the effect of LHCs on motivational direction remains elusive.

2.5 The Emotional Stroop Task (EST)

Studies presented in Chapters 3 and 4 assessed attentional bias to threat using a computerised version of the unmasked EST (Williams et al. 1996). This measures the extent to which vigilance to threat interferes with task relevant information processing. In contrast to the masked version (which examines preconscious processing), during the unmasked version stimuli are presented until a response is made allowing examination of conscious attentional processes (e.g. Mogg et al.1993). As threat-related attentional bias in anger is suggested to be confined to later processing stages (e.g. Stewart et al. 2010) it was important to use an unmasked version of the EST, to explore the UHC effects on later goal directed attention processing modulations.

Participants were shown a series of negative (e.g. '*insult'*) and neutral words (e.g. '*circle'*) presented in one of four colours, red, blue, green and white. They were required to indicate the font colour of each word as quickly and as accurately as possible while ignoring its semantic content. The EST is based on the assumption that preferential processing of the semantic content of threat words (compared to neutral words) will interfere with competing cognitive processing involved in colour responding (Mathews and MacLeod 1994). This pattern of responding, referred to as the Stroop interference effect, is indexed by slower responses to threat than neutral words. According to the parallel distributed processing model (Williams et al. 1988, 1997) words presented in the EST will be assessed according to their subjective threat value by a pre-attentive Affective Decision Mechanism (ADM). The ADM activates threat-tagged units that represent previous threat experiences and computes either a high or low threat value to the word by weighting the word against previously tagged

threat. If the word is then tagged as threatening, it activates a Task Demand Unit which priorities the processing of the threat over non-threat words. In contrast, if the word is appraised as low threat, previous resource allocation (colour responding) will be maintained without interference. When colour responses are faster for threat than neutral words, this is interpreted as a facilitation effect and has been suggested to reflect avoidance of threat (e.g. Mogg, Bradley, De Bono, and Painter 1997) or an inability to disengage attention from threat-related content to focus on task relevant responses (e.g. Fox, Russo, Bowles, and Dutton 2001).

2.5.1 EST Experimental protocol

In studies 1 (Chapter 3) and 2 (Chapter 4) a computerised version of the EST was used. This contained 80 initial neutral practice trials and 3 experimental blocks. Immediately prior to each experimental block onset, on-screen instructions prompted participants to perform UHCs, as set out by Schiff and Lamon (1994), depending on UHC condition (Study 1) or UHC group allocation (Study 2). Immediately following UHCs, the EST experimental block commenced.

Each experimental block consisted of 160 EST trials (80 threat and 80 neutral words). To minimise priming and habituation effects, trials were presented in a quasi-random order whereby no more than two words in the same colour or valence types were presented sequentially. Word stimuli were presented in red, white, blue or green (bold 20 Arial font), superimposed onto a black background. Stimuli were 52 pixels vertically and varied between 164-296 pixels horizontally (depending on word length). These were presented on

ThinkVision L200p 20.1-inch TFT LCD Monitor (6736) with a resolution of 1600 x 1200 pixels. At a viewing distance of 50 inches, words subtended approximately 0.19° of the visual angle vertically and 2.16° to 3.90° horizontally.

Each trial began with a white fixation (1000ms) followed by an average 1000ms inter-trial interval (800ms -1200ms). A threat or neutral word was then presented until either a response was made or 2000ms had elapsed. In study 1, colour responses were recorded using a button press on a standard QWERTY keyboard with corresponding coloured tabs over 'z, c, b, m' keys. In Study 2 colour responses were recorded using a RB-530 Cedrus response box 4, with corresponding coloured tabs. Stimuli presentation, RTs, error rate, and stimuli type were recorded using presentation software (www.neurobs.com, version 9.13), run on a Hewlett-Packard- HP Intel Pentium 4CPU, 2.40GHz with 256 MRAM.

2.5.2 EST Word Stimuli

The EST stimuli for Studies 1 (Chapter 3) and 2 (Chapter 4), were generated by selecting 240 threat and 240 neutral words from Affective Norms for English Words (ANEW) database (Bradley and Lang 1999). The ANEW database words are rated on valence and arousal dimensions on a scale of 1 to 9. Lower valence scores reflect more negative valenced words and high scores reflect more positively valenced words. Higher and lower scores on the arousal dimension reflect greater and lower arousal respectively. Threat words had a significantly lower valence rating (M = 2.70, SD = .75) than neutral words (M = 5.6, SD =.58), t (239) = 46.44, p < .005 and a significantly higher arousal rating (M = 5.87 SD = .91) than neutral words (M= 4.11, SD = .50), t (239) = 26.89, p < .005. Evidence has suggested

lexical differences such as word frequency and length influence colour response latencies (Larsen, Mercer, and Balota, 2006). For example lower frequency longer words result in slower word recognition and subsequently slower colour response latencies than high frequency short words. Therefore, word stimuli were matched for word length and frequency of occurrence, with all words ranging from 3-8 letters with a Kucera-Francis frequency of 1-464. 80 additional neutral words were selected for practice trials. An example of high arousal threat words included, *assault, brutal, fear* and *violent*. Low arousal neutral words included, *book, lamp, pencil,* and *paper.* The full wordlist used in Studies 1 and 2 is presented in Appendix 1.

2.6 The Dot-Probe Task (DPT)

Study 3 (Chapter 5) and Study 4 (Chapter 6) assessed attentional bias to threat using a computerised version of the unmasked DPT (MacLeod et al. 1986). As discussed previously, masked versions of the DPT only allow preconscious processing to be examined (e.g. Mogg et al. 1993). As threat-related attentional bias in anger is suggested to be confined to later processing stages (e.g. Stewart et al. 2010) it was important to use an unmasked version of the DPT to permit the exploration of the UHC effects on later conscious strategic attention processing modulations (e.g. Mogg et al. 1993).

During the DPT task, participants were presented with either a threat-neutral (T-N), positive-neutral (P-N), threat-positive (T-P) or neutral-neutral (N-N) word-pair, with each word positioned on either side of fixation. Immediately after the word-pair offset, a probe replaced either the threat (threat congruent) or the neutral word (threat incongruent) location. The use

of the term congruent and incongruent reflects the DPT literature (e.g. Koster et al. 2004; MacLeod et al. 1986) and therefore will this terminology will be adopted in this Thesis. Participants were required to respond to the probe location as quickly as possible through a button press. It is assumed that responses are faster if attention is allocated at the probe location prior to its presentation. Therefore, faster threat congruent than incongruent responding is interpreted as vigilance for threat where facilitated probe detection is assumed to reflect preferential attention allocation to the previous threat location. Conversely, slower threat congruent than incongruent responses are interpreted as an avoidance of threat (MacLeod et al. 1986).

2.6.1 Calculating DPT Bias Scores

DPT research frequently employs bias scores to explore components of attention (e.g. Bradley et al. 1998; Macleod and Mathews 1988). In accordance with previous literature (MacLeod et al. 1986), bias scores were first calculated by subtracting threat congruent responses from threat incongruent responses. Positive bias scores were assumed to reflect faster threat congruent than incongruent responses and were interpreted as vigilance/disengagement difficulties to threat words. In contrast, negative bias scores were assumed to reflect faster threat incongruent than congruent responses and were interpreted as threat avoidance. However, a major limitation of using only bias scores is that this analysis technique only compares threat congruent with threat incongruent trials and it does not provide a baseline level of attention. Without a neutral baseline to compare congruent and incongruent responses against it is difficult to identify whether the speeded responses reflect vigilance of disengagement difficulties.

2.6.2 Calculating Vigilance vs. Disengagement

Therefore, to address this and in line with a recent modification of the DPT paradigm (Koster et al. 2004), Studies 3 and 4 also included a baseline neutral-neutral (N-N) condition to delineate whether the congruency effect reflected vigilance or disengagement difficulties (Koster et al. 2004) by comparing T-N and N-N responses. Faster threat congruent than baseline responses were assumed to reflect rapid, preferential attention to threat and this was interpreted as vigilance (MacLeod et al. 1986). In contrast, slower threat incongruent than baseline responses were assumed to reflect the increased time needed to shift attention from threat to neutral locations and were interpreted as difficulty disengaging. Research that uses the modified methodology repeatedly shows that attention bias reflects difficulty disengaging from threat rather than vigilance towards it (Koster et al. 2006; Fox et al. 2004).

2.6.3 DPT experimental Protocol

Computerised versions of the DPT were used and consisted of 14 practice trials followed by three experimental blocks (RHC, LHC, and NHC). Immediately prior to each experimental block onset, on-screen instructions prompted participants to perform UHCs (described in Section 2.4 of this chapter), depending on UHC condition. Immediately following UHCs, the DPT experimental block commenced.

Each DPT block consisted of 448 trials. This was made up of 56 congruent and 56 incongruent for each word-pair (TN, PN, TP) and 112 NN baseline trials. To minimise priming and habituation effects, trials were presented in a quasi-random order so that no more than two word-pair or probe position types were presented sequentially. Words were presented in white (20 Arial font), superimposed onto a black background. Word-pairs were

26 pixels vertically and varied between 78-208 pixels horizontally (depending on word length). These were presented on a 22-inch monitor with a resolution of 1920 x 1440 pixels. At a viewing distance of 26 inches, words subtended approximately 0.4° vertically and approximately 1.5° to 4.12° horizontally.

Each trial began with a fixation (300ms), followed by a word-pair (500ms) with each word presented 2cm either side of the fixation. The inter-trial-interval varied randomly between 400 and 800ms. Immediately after word-pair offset, a probe (white dot) appeared in the congruent or incongruent position (or left and right in the NN trials) for 1500ms or until response. Participants were required to respond to probe location as quickly and as accurately as possible. In Study 3 (Chapter 4), probe location was recorded using either the Z (left of fixation) or M key (right of fixation) on a QWERTY keyboard. In Study 4 (Chapter 5), probe location (left or right) was recorded through a button on a Cedrus RB-530 response box using a left or right tab. Stimuli were presented and participants' responses were recorded, using Presentation software (www.neurobs.com, version 9.13), run on a Hewlett-Packard- HP Intel Pentium 4CPU, 2.40GHz with 256 MRAM.

In an attempt to minimise fatigue effects participants took a five-minute break between each experimental block. Latin square counterbalancing of the hand conditions was employed within participants to minimise order effects.

2.6.4 DPT word Stimuli

The DPT stimuli used in studies 3 and 4 consisted of 95 threat words, 95 positive words and 95 neutral words from Affective Norms for English Words (ANEW; Bradley and Lang, 1999). Threat words were highly negative (valence < 5) and arousing (arousal > 5), positive words were highly positive (valence > 7) and arousing (arousal > 5) and neutral words had medium valence (between 4 and 6) and were low arousal (< 5). For valence, threat (M = 2.32, SD =.40) was lower than neutral (M = 5.45, SD = .27), t (150) = -80.99, p < .05, r = *0.9;* neutral was lower than positive (M = 7.38, SD = .60), t (150) = -38.15, p < .05, r= *0.9,* and threat was lower than positive, t (150) = 81.23, p < .05, r = *0.9.* For word arousal, neutral (M = 3.99, SD =.47) was lower than threat (M = 6.15, SD =.69), t (150) = -31.33, p < .0005, r= *0.8,* neutral was lower than positive (M = 5.81, SD =.61), t (150) = -28.32, p < .0005, r = *0.8,* and positive was lower than threat, t (150) = -4.27, p < .0005, r = *0.25.* Four word-pair combinations were produced: threat-neutral (TN), positive-neutral (PN), threat-positive (TP) and neutral-neutral (NN). Based on evidence that lexical differences such as word frequency and length influence word recognition and subsequently response latencies (Larsen, Mercer, and Balota, 2006), words were matched for length and frequency, ranging from 3-8 letters, with a Kucera-Francis frequency of 1-464. Example word pairs included *bomb-cork* (TN), *cake-door* (PN) and *hate-cute* (TP). The full wordlist used in studies 3 and 4 is presented in Appendix 2.

2.7 Event-related Potential (ERP) Measures

2.7.1 Introduction

The use of the Electroencephalography (EEG) method in Study 2 (Chapter 4) and Study 4 (Chapter 6) enabled the exploration of bioelectrical potentials from on-going brain processes to be explored in relation to attentional bias. Event-related potential (ERP) measures allow precise epochs of on-going EEG activity to be time locked to the presentation of a stimulus or response (the event) within tasks such as the EST and DPT. In turn, ERPs can provide insight into the extent to which sensory and cognitive neural processes are recruited in relation to specific events.

Therefore, in Chapters 4 and 6 ERP data was presented to provide insight into underlying cognitive processes associated with UHC modifications in threat-related attentional bias in the EST (Chapter 4) and DPT (Chapter 6). The advantages of using ERP methods in the study of attentional bias over other neuroscience procedures is that EEG/ERP is non-invasive, relatively cost effective and provides millisecond temporal resolution that reveals transient changes in brain activity that promote specific cognitive functions. However, when compared to Functional Magnetic Resonance Imaging (fMRI) and Positron Emission Tomography (PET) that localise brain activity with millimetre precision, EEG/ERP can only localise with centimetre precision. This low spatial resolution is caused by three main issues; 1] a highly conductive skull, cerebrospinal fluid and extra-cranial tissue distort electrical field brain topography; 2] The dendritic activity reflected in EEG does not reflect activity from deep brain structures; 3] The orientation of activated cortical columns influences the dispersion of signals on the scalp. For example, when perpendicular to the scalp powerful signals are generated but perpendicular columns of the same polarity, at other brain regions, can inflate this signal. When cortical columns are parallel to the scalp activity is often projected to sites

other than those directly above it. This is referred to as the inverse problem and can be a major limitation of ERP studies. However, issues pertaining to poor spatial resolution can be somewhat minimised with Source Localisation or Dipole Source techniques that apply algorithms to improve spatial resolution (See Grech et al. 2008, for a review).

Despite the relatively poor spatial resolution capabilities of EEG when compared to fMRI, that suffer from poor temporal resolutions (seconds), EEG/ERP has a far superior temporal resolution (milliseconds). This can reveal transient changes in brain activity that reflects specific underlying neural processes. As such, the ERP method enables the development of theories about how brain mechanisms promote specific functions that occur during attentional bias.

A major advantage of using ERPs it that it allows specific brain signals that are phase-locked to an observable event (signal) to be extracted from the ongoing non phase-locked EEG (noise) that they are embedded (Luck 2005; Burgess 2012). ERPs improve the signal to noise ratio (SNR) by time locking an evoked brain signal that exists in the on-going EEG and averaging it over numerous trials (Burgess 2012). The averaging process is based on the assumption that the time locked brain activity (signal, +/- 5 μV) will be the same in every trial but that the general electrical 'noise' (signal, +/- 50 μV) will vary randomly (Rugg and Coles, 1995). As such, the 'noise' is averaged to zero while the signal of interest is isolated and extracted in a resultant waveform known as the ERP. The following sections discuss procedures used to remove this noise.

2.7.2 Cleaning Raw Data

Another important consideration when using the ERP method is that 'noise' created outside the brain needs to be removed when extracting ERP data. This 'noise' can be of physiological origin such as eye movements (e.g. blinks, vertical and lateral movements), frontalis and temporalis muscles movement (e.g. jaw clenching), and tongue movement (e.g. chewing or sucking) or of extra-physiological origin including laboratory equipment, video monitors, and AC lines (see, Usakli 2010)

2.7.2.1 Filtering

Extra-physiological artifacts can be removed by filtering, prior to averaging, the electrical signal to the frequency of interest in cognitive neuroscience research, typically between 0.01 to 40 Hz. AC line artifacts (50Hz) and video monitor refresh (60-75 Hz) can be removed from the raw signal with low pass filtering techniques. This allows low frequencies to pass, but diminishes high frequencies from the signal. While an in depth discussion on filtering is beyond the scope of this Thesis it is important to note that different filtering techniques influence later waveform processing interpretations drawn. Therefore, a good understanding is needed when applying filters to ERP data (see Luck 2005).

2.7.2.2 Artifact Rejection

In order to remove artifacts such as eye movements, artifact rejection or correction techniques are employed prior to averaging. One such method is the automatic artifact rejection technique found in the Brain Electric Source Analysis software (BESA; Scherg and Berg 1990). This method removes all trials that contain eye movement above maximal predetermined amplitude (e.g. 100 μV). A major issue with this method is that it reduces the

number of trials available for exploring time and frequency domains of the EEG and reduces the signal to noise ratio of the data. Another issue with this technique is that eye movements are often time locked to stimuli or behavioural events, meaning that rejecting all trials containing eye movements would result in the removal of most trials. While suitable for on-going frequency analysis approaches this approach is not well suited for research where the ERP may often be time locked to the artifact.

As discussed previously, ERP analysis is based on the assumption that artifacts will vary randomly across trials and average to zero. If non-random eye movements are not removed as artifacts, they will be averaged into the ERP waveform leading to misinterpretation of the data. This issue can be resolved by applying an artifact correction algorithm in BESA that subtracts the predicted influence of artifact related fluctuations from the ERP (Berg and Scherg 1994 see also Ille, Berg, and Scherg 2002). This method ensures that maximum trial numbers are accepted for averaging, that a good SNR is achieved and that eye movements do not corrupt the data. For this reason, artifact correction algorithms were used to remove eye movements in the ERP studies within this Thesis.

2.7.3 The ERP Waveform

2.7.3.1 Importance of Pre-Stimuli Baselines

ERPs waveforms reflect voltage changes across time and are measured as the difference between brain activity at baseline (usually recorded during pre-stimuli onsets of 100-200ms) and the event. Comparing the event-related potential to a baseline measure is important when decomposing an ERP waveform. The baseline activity is gained by averaging activity over this pre-stimulus interval. It is assumed that brain activity at this point in time is random and not influenced by an event: as such it will be averaged to zero. However, it has been

suggested that if this baseline measure is not sufficiently long enough (>100ms) then activity from the previous event will introduce noise into the baseline which in turn will introduce noise in the ERP itself (Luck 2005).

2.7.3.2 ERP Component Characteristics

Another important aspect of ERP research is describing the ERP component itself. One classification for labelling components is based on measures of peak polarity (i.e. the most negative or positive deflections in the wave), amplitude, latency (often measured as time from stimuli onset), and scalp distribution. According to the order approach, labelling is based on the sequence that the peak occurs and its polarity. For example, occipital P1 refers to the first positive on-going peak in the waveform occurring at occipital sites and frontal N2 reflects the second negative on-going peak arising at frontal sites. In contrast, the latency approach identifies a component by its polarity and by the latency since stimuli onset. For example, P100 refers to a positive peak that occurs 100ms post stimuli onset and N200 reflects a negative peak that occurs 200ms after stimuli onset. These objective labelling systems can be effectively applied to early components although applying peak-naming conventions to later components (e.g. P300 and N400) that are not typically identifiable by their specific peaks is somewhat problematic. Furthermore, these labelling systems are meaningless concerning ERP functionality and psychological interpretation (Luck, 2005).

An early functional definition devolved ERP waveforms into short (<100ms) or long (>100ms) latencies, to reflect exogenous (e.g. evoked sensory processing) and endogenous (e.g. information processing) components respectively (Donchin, Ritter, and McCallum 1978; Donchin 1981). However, this definition was suggested to be overly simplistic after findings

emerged that showed exogenous and endogenous characteristics existed in both early and late components, depending on stimulus properties (e.g. Shibasaki and Miyazaki 1992). As a result, functional descriptions of ERPs are now classified in terms of specific cognitive processes and their neuroanatomical generators (e.g. Naatanen and Picton 1987). This definition can be valuable when identifying peaks that occur at identical times but are localised at electrode sites related to different cognitive processes. In line with this definition, the ERP component has more recently been defined by Luck (2005) as "scalp recorded neural activity that is generated in a given neuroanatomical module when a specific computational operation is performed" (p 59).

2.7.3.3 Amplitude Measures

Identifying which aspect of the ERP (amplitude, latency, and generator) is the most appropriate for addressing the research question is an important consideration when conducting ERP analysis. While ERP amplitudes are suggested to reflect the strength of cognitive engagement, latencies reflect the time course of processes under investigation (Luck et al 2000). It is important to note that the ERP studies presented in this Thesis used amplitude measures to explore research questions. Therefore, for brevity discussion of latency measures are not included.

Two main amplitude measures are applied to the decomposition of the ERP, peak amplitude and mean amplitude. Peak amplitude can be isolated in to simple peak amplitude and local peak amplitude. Simple peak amplitude is measured at the point, in a predefined time window, when the localised brain activity reaches maximum voltage. It has been suggested that this method is extremely susceptible to issues of overlapping components when the peak is at the edge of the time window (Luck, 2005). Local peak amplitude in contrast, is

measured by identifying a maximum peak that has three to five smaller peaks at either side of it. It has been argued that this measure is less susceptible to overlapping components at the edge of the time window (Luck 2005).

Peak amplitude analysis has been suggested to be problematic when applying it to components that have a flatter morphology and no well-defined peak such as the P300 and N400 (Luck 2005). Indeed, Luck (2005) went further and argued that there is nothing special about the point in time that a maximum voltage is reached and that any conclusion drawn from using this measure would be highly distorted by noise and in some instances component overlap. Given this potential source of ambiguity a strong argument could be made that peak analysis should only be used for early, and not late, ERP component analysis.

A more appropriate measure of amplitude is mean amplitude derived by calculating the mean voltage over a defined time window. Mean amplitude measures can be used to identify voltage differences between two conditions, with greater voltage indicating greater overall neural engagement in one condition compared to another. Mean amplitude has a number of strengths when compared to peak measures. Firstly, a more narrow time window can be used for mean amplitude, making this measure less susceptible to noise from overlapping components (however, it is important to note that if the latency of a component varies across a condition then the mean value may still be sensitive to overlapping components). Secondly, because mean amplitude measures take a range of time points rather than a single point (as in peak measures) it is less sensitive to high frequency noise. Given the strengths of this amplitude measure, the ERP research within this Thesis will use mean amplitude for later components, namely the P3b and the negative slow wave NSW. In contrast, for earlier P2 and N2 components peak amplitudes were used.

2.7.4 ERP Indices of Attentional Bias

In this section an overview is provided of ERP components that are related to attentional processing and are therefore central to this Thesis. While previous literature use a wide range of components to explore sensory and cognitive aspects of attentional processing at different neural locations for brevity only ERP components that are explored in studies 2 and 4 are discussed.

2.7.4.1 P2 Component

In Study 4 (Chapter 6), modulations of the frontal-frontocentral P2 ERP (maximum amplitude around 200–250 ms following stimuli onset) were explored in relation to valence and UHCs in the DPT. The P2 denotes rapid detection, low-level categorisation of stimuli (Eimer and Holmes 2007), the phonological processing of words (Rugg and Coles 1995) and early global affect evaluation that guides approach and withdrawal motivated behaviour (Schapkin, Gusev and Kuhl 2000). Research has also shown that P2 amplitude is influenced by valence albeit with inconsistencies. For example, some have shown that P2 is enhanced for threat only (Huang and Lou, 2006), others for positive stimuli only (Schapkin et al. 2000) and other have shown this effect for both threat and positive stimuli alike (Herbert, Kissler, Junghoffer, Peyk, and Rockstroh 2006; Carretie, Martin-Loeches, Hinojosa, and Mercado 2001; Carretie, Hinojaosa, Martin-Loeches, Mercado, and Tapia 2004). These inconsistencies may reflect methodological differences such as stimuli type (words vs. faces) and individual differences in key personality characteristics. However, as P2 has been shown to denote evaluative processing of emotional words in healthy samples (e.g. Schapkin, et al 2000; Trauer, Kotz and Müller, 2015), Study 4 utilised this ERP component to explore whether UHCs influenced early effective evaluation of threat.

2.7.4.2 N2 Component

N2 a negative deflection that occurs over frontocentral regions at 200 to 350ms following stimuli onset, is thought to reflect changes in controlled, effortful, and conscious processes (Mangun and Hillyard 1995; van Veen and Carter 2002). The N2, which has been found to be generated by the anterior cingulate cortex during conflict (ACC; e.g. Banich 2009; van Veen and Carter 2002; Kanske and Kolz 2007), denotes the extent that attentional control is recruited to resolve conflict and inhibit incorrect responses (Braver, Barch, Gray, Molfese, and Snyder 2001; Jones, Cho, Nystrom, Cohen, and Braver 2002; Nieuwenhuis et al. 2003; van Veen and Carter 2002). Furthermore, N2 is thought to reflect a "gating" mechanism whereby motivationally relevant information such as threat gains access to cognitive control systems (Dennis and Chen 2007; Dennis 2009). For this reason, N2 provides a neural basis for emotion-cognition integration and was deemed appropriate for studying the effects of UHC on influencing top down attentional processing in Studies 2 and 4 in this Thesis.

Previous research has shown larger N2 for threat than neutral stimuli in relation to high anger (Stewart et al. 2010). In contrast, healthy participants have been found to elicit smaller N2 for threat than for positive or neutral words (Perez-Edgar and Fox 2003). According to neural efficiency theories, reduced activity during conflict indicates greater efficiency of resource allocation during task performance (Dennis and Chen 2007; Gray, 2004; Gray, Braver, and Raichle 2002). Furthermore, the reduced N2 found for threat in healthy participants is consistent with the dual system theory of cognitive control (Botvinick, Braver, Barch, Carter, and Cohen 2001; Botvinick, Cohen, and Carter 2004). This model posits that, inhibition and cognitive control resources are divided between monitoring and response processes to promote attentional performance. As such, enhanced N2 amplitudes for threat in high anger

indicate that more resources were allocated to action monitoring at the detriment of attentional performance.

2.7.4.3 P3b Component

The P3b is possibly the most researched of the ERP components. This positive deflection occurring at 300-600ms following stimuli onset is predominantly characterised by a parietal distribution. The P3b is suggested to be mainly insensitive to sensory processing and instead is thought to reflect stimuli evaluation to promote task relevant responding (e.g. Nieuwenhuis et al. 2005; Duncan et al. 2009*).* Amplitude fluctuations of this component have been found to be related to conscious processing (e.g. Donchin and Coles 1988) increased resource deployment, stimuli evaluation, and attentional allocation (e.g. see Polich 2007 for a review). Associated with top down attention and stimuli significance, the P3b has been shown to be larger for target than non-target stimuli (e.g., Johnson 1986; Duncan-Johnson and Donchin, 1977). The P3b is also sensitive to bottom-up emotionally significant information with larger amplitudes for emotionally relevant compared to neutral stimuli (e.g. MacNamara et al. 2009; Eimer and Holmes 2002). This component also provides a means to explore the extent that threat processing has influenced competing cognitive processing. For example, smaller P3b for threat compared to neutral targets denotes increased task difficulty and shows threat interfered with target responding (Duncan-Johnson and Donchin 1977; MacNamara, et al. 2009).

As discussed in Chapter 1, attentional bias research has shown high anger is related to larger P3b for threat than neutral words (Stewart et al., 2010). These findings provide further evidence that threat receives priority processing and greater allocation of attentional resource than non-threat words in relation to high anger. However, others have shown that high anger

is related to preferential processing of emotional over neutral information regardless of valence, indexed by enhanced P3b for emotional compared to non-emotional stimuli (Bertsch et al. 2009).

Taken together, evidence suggests P3b is larger for motivationally significant information, regardless of whether the information is task relevant or the emotional content of the information implies relevance, as in the case of a natural target such as threat (MacNamara et al. 2009). For this reason, P3b was used in Study 2 and 4 to provide an index of how UHCs and trait motivational tendencies influence the motivational significance of threat.

2.7.4.4 Negative Slow Wave (NSW) Component

The NSW is negative going deflection localised to frontocentral sites at between 400 – 500 ms post-stimuli onset and is suggested to reflect activation of the ACC (Liotti et al. 2000; Veen and Carter 2002). The NSW is suggested to provide a neural index for conceptual level inhibition and efficiency during conflict, with larger NSW for threat compared to neutral words denoting threat-related interference (West 2003; West and Alain 2000; van Hooff, Dietz, Sharma, and Bowman 2008 Markela-Lerenc, Ille, Kaiser, Fiedler, Mundt, and Weisbord, 2004). The NSW provides an index of the efficiency of inhibitory mechanisms used to supress threat conflict in the EST and is therefore appropriate for studying the effects of UHCs and anger on attentional control processing in the Study 2.

2.8 EEG Data Collection

2.8.1 Preparation of Participants

Participants were prepared for EEG by firstly measuring the circumference of the head immediately under the eye brow and above the inion, to identify the cap size needed for each

participant. Once the cap size was determined the distance between the inion and nasion as well as the distance between the left and right pre-auricular points was recorded and divided by two. This procedure enabled the identification of the Cz electrode location to ensure that the cap was positioned in the correct place in relation to the 10-20 system. Once the cap was fitted these measurements were taken again to ensure that Cz was at the half way point between the inion and nasion and the left and right pre-auricular points.

Following the fitting of the cap, horizontal electro-occulargram (EOG) positions the epicanthus of the right and left eye, and vertical positions, the supra-orbital and infra-orbital positions of the right eye were cleansed. Biosemi flat type, touch-proof electrodes with sintered Ag-AgCl electrode pallets, were prepared with conducting gel and applied with adhesive disks and surgical tape to the cleansed horizontal and vertical EOG sites. Following the application of EOG electrodes, preparation of EEG sites were conducted. This involved gently moving the hair from the electrode sites with a Q-tip until the scalp was visible and applying Signa conductive gel to the EEG sites with a needleless syringe. The Signa gel provided a contact between the skin and the electrode with low impedance. 32 'pin type' silver-silver chloride (Ag-AgCl) active electrodes were then mounted onto the Biosemi lycra cap.

2.8.2 EEG Data Acquisition

In studies 2 and 4, EEG data were recorded using a 32-channel EEG with silver-silver chloride (Ag-AgCl) 'pin type' Biosemi active two electrodes referenced to the common mode sense (CMS); active electrode, (BioSemi Active-Two, BioSemi Amsterdam). A major benefit of using active electrodes is that each electrode provides an impedance transformation which prevents significant interference voltages from being generated, making impedance

level unimportant. Electrical brain activity was recorded from Fp1, Fp2, Pz, Fz, O1, O2, P3, P4, P7, P8, C3, C4, T7, T8, F3, F4, F7, F8, Cz, Oz, CP5, CP6, CP1, CP2, FC1, FC2, FC5, FC6, AF3, AF4, PO3 and PO4 sites according to the international 10-20 system. BioSemi flat type, touch-proof electrodes with sintered Ag-AgCl electrode pallets, at horizontal and vertical electro-occulargram (EOG) positions, allowed eye movements to be recorded for later artefact correction. The quality of electrode contact was identified and controlled by ensuring each electrode fell between -25mV and 25mV, using the BioSemi DC offset. Signals were band pass filtered between 0.01-100 Hz, with a sampling rate of 512Hz and a bandwidth of 104Hz. Signals were digitised with a 24-bit Active Two BioSemi A/D converter. Trial events, such as word type and colour (Study 2) and word-pair, probe position (Study 4) and onset/offset times were generated by presentation (www.neurobs.com, version 9.13). These events were synchronised with triggers and transmitted to the BioSemi data recording system through a standard parallel port that recorded EEG signals, concurrently.

2.9 Offline ERP Pre-Processing

Processing was performed using BESA software (version 5.1.8, MEGIS Software GmbH, Gräfelfing, Germany). All electrodes were referenced to a common average reference. All raw signals were filtered between 0.01Hz and 30Hz prior to averaging. This procedure ensured that sinusoidal oscillatory artifacts related to AC line noise and spiky artifacts, time-locked to video monitor refresh, were excluded from analysis.

Each participant's data was manually inspected for blinks with a typical topography, characterised by a monophasic deflection (50 – 100μV) in the fronto-polar regions at 200-400ms. An artefact correction procedure based on Berg and Scherg (1994, see also Ille et al. 1997, 2002) spatial component method (principle component analysis) was performed to

subtract the estimated contribution of blinks, related fluctuations, away from the signal. To ensure that all trials were artifact free, an artifact rejection scan was also carried out on the data. Epochs were then created from a pre-stimuli baseline start position of -200ms to 700ms post stimulus end position for each participant. All epochs underwent baseline correction prior to averaging.

Participant's data were then processed to create averages for each condition. Data were then combined to create a grand average data set for all target conditions. In Study 2, four grand averages were produced: RHC threat words, RHC neutral words LHC threat words and LHC neutral words. In Study 4, six grand averages were produced; T-N congruent, T-N incongruent, P-N congruent, P-N incongruent, T-P congruent, and T-P incongruent.

Once grand averages were produced, a visual inspection of the grand ERP waveforms was performed and previous literature was consulted (e.g. Stewart et al. 2010; van Hoof et al. 2008 Bar-Haim et al. 2005; Eldar and Bar-Haim 2010; O'Toole and Dennis 2012) to select electrode positions and components for analysis.

In Study 2 (Chapter 4), electrode positions and scoring epochs of N2 (137-223ms), P3b (340-580ms) and NSW (330-700ms) were selected based on previous EST ERP literature (e.g. Thomas et al. 2007; Stewart et al. 2010; van Hoof et al. 2008). The N2 peak amplitude was recorded at frontal (F3 and F4), frontocentral (FC1 and FC2), and central (C3and C4) electrode sites. The P3b mean amplitude was recorded at centroparietal (CP1 and CP2), parietal (P3 and P4), and parietooccipital (PO3, and PO4) sites. The NSW mean amplitude was recorded at frontal (F3 F4), frontocentral (FC1 and FC2), and central (C3 and C4) sites.

In Study 4 (Chapter 6), electrode positions and scoring epochs P2 (190-260 ms), N2b (250-310 ms), and P3b (290-550 ms) were selected following visual inspections of grand ERP waveforms and with reference to previous DPT ERP literature (e.g. Bar-Haim et al. 2005; Eldar and Bar-Haim 2010; O'Toole and Dennis 2012). The P2 peak amplitude was recorded at frontal (F3 and F4) and frontocentral (FC1 and FC2) electrode sites. The N2b peak amplitude was recorded at frontal (F3 and F4), frontocentral (FC1 and FC2), and central (C3and C4) electrode sites. The P3b mean amplitude was recorded at centroparietal (CP1 and CP2), parietal (P3 and P4), and parietooccipital (PO3 and PO4) sites.

2.10 Data Analysis

Statistical analyses were carried out using IBM SPSS Statistics for Windows, Version 22.0. A series of Analysis of Variance (ANOVA), Analysis of Covariance (ANCOVA), and t-tests were conducted to test the predictions made in this Thesis. Bonferonni corrections were applied for multiple comparisons. Statistical analyses employed a two-tailed alpha level of .05 and effect sizes of significant results are reported as proportion of explained variance (partial eta squared [ηp^2]).

While ANOVA is based on the assumption that data will be normally distributed and the variance of the different conditions are equal, this analytical technique is robust when violations are mild to moderate (Luck, 2005). As ERP studies used a within subject variable of electrode position, with three levels, the assumption of homogeneity of covariance is required. Homogeneity of covariance assumes that the degree of correlation between electrode position 1 and electrode position 2 is equal to the degree of correlation between electrode positions 2 and 3. However, as data from contiguous electrodes is typically more

correlated than data from more distant electrodes, homogeneity of covariance is often violated in ERP studies (Luck, 2005). Violations of this nature will lead to artificially low p-values. This inflation of a type 1 error can be resolved by applying the Greenhouse-Geisser epsilon adjustment (see Jennings and Wood, 1976). Luck (2005) has suggested that when more than two electrode position are used in an ANOVA that, reporting Greenhouse-Geisser adjustments are essential. As such, all ERP results will be reported using the Greenhouse-Geisser adjustment.

CHAPTER 3

STUDY 1: THE RHC EFFECTS ON ATTENTIONAL BIAS TO THREAT

3.1 Introduction

The aim of the study presented in this Chapter was to investigate whether right hand contractions (RHCs) would modulate attentional processing of threat words among healthy participants. As discussed in Chapters 1 and 2, RHCs prime approach motivation (Harmon-Jones 2006) and increase anger-related behaviour (Peterson et al. 2008) in healthy samples. Given this evidence and in line with theories that posit emotion regulation is influenced by attentional processes (Gross 1998, 2005), it was predicted that RHCs would increase approach motivation and anger by modulating attentional bias to threat.

Exploring anger-related attentional bias to threat is important for a number of reasons. First it has been suggested that emotions such as anger occur when an individual attends to information and evaluates it as important to a current goal (Gross and Thompson 2007). Secondly, as discussed in Chapter 1, attentional bias to threat has been well-documented in high state and trait anxiety (for review see, Bar-Haim et al. 2007) but as discussed in Chapter 1, only a handful of studies to date have explored attentional bias to threat in relation to high trait and state anger (e.g. Smith and Waterman 2003; van Honk et al. 2001). Third, the lack of research is perplexing given the effects that anger has on individuals and society in general (e.g. Bettencourt et al. 2006; Williams et al. 2000). See Chapter 1, for a discussion about the interpersonal, health and social effects of chronic levels of high trait anger. Furthermore,

attention processing of threat information is well known to play an important role in automatic anger regulation (Gross 1998; Gross and Thompson 2007). Compared to more deliberate forms of emotion regulation such as suppression, that are costly in terms of conscious effort, modulating attentional components of emotion regulation may provide a relatively effortless way to manage anger (Gross and Thompson 2007). Indeed, a wealth of evidence is emerging to show ABM programmes provide a successful treatment for reducing other negative effects such as anxiety (e.g. Heeren, et al. 2015). As such, gaining a better understanding of attentional processes underlying anger could have practical implications for informing efficient anger reduction interventions in people susceptible to anger (Meier et al. 2006).

As discussed in Chapter 1, previous research indicates that individuals with high anger show a preference for processing threat compared to neutral information and this then interferes with competing cognitive processes important for EST performance (e.g. Smith and Waterman 2003). Details of the EST method are presented in Chapter 2. Research also suggests that anger-related interference happens at later attentional stages (e.g. van Honk, Tuiten, van den Hout et al. 2001). These findings lead to the assumption that high anger individuals may be unable to employ later goal directed control over attentional bias to threat. While previous evidence provides important insight into the relationship between anger and attentional bias to threat, its correlational nature has led to uncertainty about the causal relationship between anger and attentional bias to threat. These ambiguities may be somewhat resolved experimentally by exploring how manipulations of anger-related brain networks affect attentional bias to threat-related stimuli.

As also discussed in Chapter 1, EEG research has highlighted that high anger, a negative approach motivated emotion, is related to greater relative left frontal brain activity (for a review see, Harmon-Jones, Gable, and Peterson 2010). However, much of the evidence supporting this view is correlational and does not explore anger-related frontal brain asymmetry in the context of cognitive components of emotion regulation. This is perplexing given that left PFC is known to be important for cognitive processes that promote the regulation and control of emotional and motivational goal responding (see Davidson 2003; 2004 for reviews).

Therefore, it is possible that high anger may be related to increased left frontal brain activity due to the greater need to inhibit anger based responding. The dissonance between attentional bias and frontal brain asymmetry literature has led to gaps in understanding about how differential patterns of hemispheric activity influence threat-related attentional bias. This has prompted the question of how anger-related left frontal brain activity is implicated in attentional bias. Taken together, the association between anger, motivation, cognitive control and frontal activity suggests that the left frontal regions may play a role in attentional bias to threat.

However as discussed in Chapter 1, only one study to date has explored the hemispheric specialisation of attentional bias and this showed that greater left DLPFC activation resulted in approach-motivated attentional bias towards threat, while greater right DLPFC activity produced avoidance. (d'Alfonso et al. 2000). The positive association between left frontal activity and anger may reflect an inability to inhibit threat-related attentional bias (e.g. van Honk et al. 2001). Importantly, this challenges the view that greater DLPFC activity is associated with increased cognitive control (Davidson 2004). As such further research is

needed to explore whether increased approach motivation and anger-related frontal brain asymmetry modulates attentional bias to threat.

The UHC method (Schiff and Lamon 1994, also see Chapters 1 and 2), has been shown to reliably increase DLPFC activity (Harmon-Jones 2006) and provides a method to modulate left frontal brain activity. Research has found that RHCs prime approach motivation (Harmon-Jones, 2006) and increase aggression (Peterson, et al., 2008) in healthy samples. These findings support EEG observations that increased left frontal activation is positively associated with approach motivation (see Harmon-Jones, 2003 for a review).

While the UHC method has been shown to modulate emotional and motivational behaviours (Harmon-Jones 2006; Peterson et al. 2008), it is uncertain which antecedent-focused components of emotion regulation were modulated to increases anger. As discussed in Chapter 1, emotions can be regulated at situational selection, situational modification, attentional deployment, and appraisal stages before experiential, behavioural, or physiological responses occur (Gross 1998, 2001, 2002). Given that the situation was controlled in the Peterson et al. (2008) study it can be assumed that the increased anger behaviour following RHCs was a result of modifications in attentional deployment or appraisal stages of emotion regulation. Methodologically, attentional deployment can be operationalised and measured more objectively than appraisal. Furthermore, evidence has consistently shown that attentional bias to threat is important in the aetiology and maintenance of anger-related emotion disorders. For this reason, an investigation of the effects of the RHC on attentional bias to threat was warranted.

The RHC method is now considered a reliable method for inducing aggression and approach motivation (Peterson et al. 2008; Harmon-Jones 2006). Furthermore, studies that have employed this method to explore the effects of increased left frontal brain activity on emotional and motivational responding have provided support for motivational models of frontal brain asymmetry. However, the findings gained in this research are consistent with both motivational and cognitive control views of frontal brain asymmetry. Specifically Peterson et al. (2008) showed that inducing greater left frontal brain activity via the RHC will increase aggression towards the source of provocation. According to motivational models (Harmon-Jones 2003), RHCs would activate networks associated with approach motivational action tendencies, which then cause increased aggression in response to insult. According to cognitive control models (e.g. Davidson 2003; 2004; Miller and Cohen 2001) RHCs would modulate circuits that control anger processing in order to facilitate goal directed behaviour. However, goal directed behaviour in this instance may have been to remove the source of provocation. As such it is unclear how increased left frontal brain activity modulates cognitive control of emotional responding. Exploring cognitive processing in the presence of threat may shed light on this question.

To date no research has examined the effects of UHCs on the cognitive processing of emotional stimuli. Therefore, Study 1 aimed to investigate the behavioural effects of RHCs compared to LHCs and NHCs on attentional bias to threat (interference/facilitation) compared to neutral words using a modified EST (Williams et at. 1996). The design of this Study differed from previous correlational attentional bias research (e.g. Smith and Waterman 2003), in that it attempts to modulate attentional bias through the use of the UHC method. In doing so, Study 1 attempts to gain insight into the causal direction of the

relationship between anger-related approach motivation and attentional bias to threat using the UHC method.

In addition, while research does exist that has explored the causal relationship between left frontal brain activity and attentional bias to threat (d'Alfonso et al. 2000) the rTMS method used in past research is subject to methodological issues that are not associated with the UHC method. For example, while the safety of rTMS continues to be supported by published literature there is evidence that this method has induced seizures in a handful of participants (for a review see, Rossi, Hallett, Rossini, and Pascual-Leone 2009). Furthermore, evidence also suggests that rTMS can be painful inducing headache and neck ache, depending on individual predisposition, as well as intensity and frequency of stimulation (Rossi et al. 2009). As participants are warned about these possible side effects, this may cause state anxiety, confounding the findings of studies exploring the effects of brain activity modulation on emotional processing. For this reason the UHC method provides a less invasive and more reliable technique of modulated frontal brain asymmetry on attentional bias to threat. As such the Study presented in this Chapter provides the first non-invasive exploration of the effects of induced changes in frontal brain asymmetry on attentional bias.

Given that the UHC method has not been explored previously in relation to attentional bias modification the main aim of the Study was to identify whether the UHC would effectively modulate attentional bias to threat. The present Study also aimed to provide insight into whether the emotion and motivational effects found for RHCs in previous research (e.g. Peterson et al. 2008) were a consequence of modulations of attentional bias. It may also shed light on how RHC induced anger-related frontal brain asymmetry patterns influence the

attainment of goal related cognitive processing when competing threat-related information is present.

3.1.1 Predictions

Previous literature that has shown that attentional bias to threat is related to approach motivated high anger (e.g. van Honk, Tuiten, de Hann et al. 2001) would suggest that RHCs will increase attentional bias towards threat (an EST interference effect). However, evidence suggesting greater left frontal activity is positively associated with increased cognitive control and approach motivation (e.g. Harmon-Jones 2006; Davidson 2003; 2004) would suggest that RHCs would improve task related performance (an EST facilitation effect). While it can be predicted that attentional bias to threat will be modulated following RHCs, the exploratory nature of this Study means that the modulatory direction of the RHC effects remain unclear.

As such, it was hypothesised that:

1. There will be no main effect of UHC or word-type on EST RTs
2. RHCs compared to LHCs and NHCs will modulate attentional bias to threat compared to neutral words by either:
    a. Increasing RTs to threat compared to neutral words (interference), reflecting increased anger and approach motivation towards threat.

       Or

    b. Reducing RTs to threat compared to neutral words (facilitation), reflecting increased cognitive control over attentional bias to threat in favour of task performance.

3.2 Method

3.2.1 Design

The Study employed a 3 (UHC; right hand (RHC), left hand (LHC), no hand-contractions (NHC) x 2 (word-type: threat, neutral) repeated measures design. RTs (ms) to threat and neutral words provided an index of attention bias to threat.

3.2.2 Participants

Participants were an opportunity sample of 30 healthy Psychology students from Coventry University. The sample consisted of 24 females (aged; *M* = 19.21, *SD* = 1.14) and six males (aged: *M* = 18.60, *SD* = .89). Participants met the inclusion criteria (see Chapter 2). Coventry University Ethics Committee approved the current study and written informed consent was gained from each participant.

3.2.3 Materials

3.2.3.1 Word Stimuli

Information about the generation of the EST words can be found in Chapter 2, Section 2.5.1.2. The words used in this Study are also presented in Appendix 1.

3.2.3.2 Emotional Stroop task (EST)

Information about the EST protocol can be found in Chapter 2, Section 2.5.1.1.

3.2.3.3 The UHC method

Information about the UHC procedure is presented in detail in section 2.4.

3.2.4 Procedure

Before the EST, participants provided informed consent, demographic information and completed the EHI-r (Williams, 1986). Participants carried out the EST study over three separate sessions running over three consecutive weeks at the same time of day and on the same day of the week. This was done to ensure that there were no carry over effects, such as practice or fatigue. Latin squared counterbalancing dictated which condition (RHC, LHC, and NHC) would be performed during each session and standardised verbal instructions on how to complete the designated hand contraction were provided. During the EST, participants responded as quickly and accurately as possible to the font colour presented through a button press (See section 2.5.1 for details). Each session lasted 40 minutes, with 5-minute breaks between blocks to minimise fatigue effects.

3.2.5 Data preparation

Prior to analysis, incorrect and missed colour naming responses, (1.5% of total responses), were eliminated. Trials with RTs < 200ms and > 3SDs above each participant's mean (2.7% of total responses) were also removed (cf. Van Honk, Tuiten, de Haan et al. 2001). All outliers in this range were replaced by participant's mean RTs for that condition. After data screening, one participant's data were excluded due to excessive missing and erroneous responses. Therefore, the final analysis consisted of 29 datasets. An alpha level of 0.05 was used for all statistical tests unless stated otherwise. Bonferroni corrections were applied and significant interactions were explored using repeated measure contrasts.

3.3 Results

3.3.1 Data analysis

To assess the effect of UHC on attention bias to threat words, mean RTs (ms) were entered into a 3 (RHC, LHC, NHC) × 2 (word-type; threat, neutral) repeated measures analysis of variance (ANOVA). Mauchly's test indicated that sphericity was assumed for hand condition, χ^2 (2) = .35, $p > .05$ but was violated for the hand × word interaction, χ^2 (2) = 9.20, $p < 0.05$. Therefore, degrees of freedom were corrected (ε= .78) using Greenhouse-Geisser estimates of sphericity for the hand × word interaction.

Summary data showing participants mean RTs to threat and neutral words in all conditions are shown in Table 3.1. These indicated that in the NHC condition participants responded slower to threat than neutral words. In contrast in the RHC and LHC conditions participants responded faster to threat than neutral words.

In line with prediction 1, the ANOVA revealed no main effect of word type, i.e. there was no RT difference between threat and neutral words, F (1, 28) = 3.30, $p > 0.05$, η_p^2 = .11, and no main effect of UHC condition, F (2, 56) = 1.74, $p > 0.05$, η_p^2 = .06. In support of prediction 2, the ANOVA revealed a significant UHC condition × word-type interaction, F (1.55, 43.45) = 3.77, $p < 0.05$, η_p^2 =.12. Planned contrasts revealed no significant differences between threat and neutral words in the NHC, F (1, 28) = 1.01, p = .32, η_p^2 = .04 or the LHC conditions, F (1, 28) = 2.66, p = .11, η_p^2 = .09. However, contrasts did reveal that RTs were significantly faster for threat than neutral words in the RHC condition, F (1, 28) = 6.52, $p <$ 0.05, η_p^2 = .19. This illustrated that RHC was related to a facilitated EST responding in the presence of threat compared to neutral words.

Table 3.1. Mean EST RTs (ms) for threat and neutral words in the RHC, LHC and NHC conditions are shown for 29 participants. Standard deviations are shown in parenthesis

Hand condition	**Threat words** *M (SD)*	**Neutral words** *M* **(SD)**
RHC	815.47 (166.78)	832.77 (170.44)
LHC	834.89 (159.43)	837.00 (156.53)
NHC	806.81 (168.54)	803.37 (164.38)

3.4 Discussion

This study provides some of the first evidence that RHCs induced threat-related attentional bias in the modified EST which may reflect increased cognitive control or approach motivational task responding. In line with prediction 1, findings revealed no main effect of word type, and no main effect of UHC condition, on RTs. Prediction 2b, that RHCs but not LHCs or NHCs would induce attentional bias to threat (producing faster RTs to threat compared to neutral words), was also supported. The results showed a facilitation effect, only in the RHCs. No attentional bias effects were observed in the other two conditions.

This pattern of results support evidence for the positive association found between left frontal activity and attentional bias to threat (d'Alfonso et al. 2000; Eckhardt and Cohen 1997; Van Honk et al. 2001). However, the facilitation effect found in the RHC condition directly conflicts with the argument that left brain frontal activity is causally related to interference for threat (d'Alfonso et al. 2000). Although speculative we can infer that the facilitation

effect observed with the RHCs may reflect greater recruitment of cognitive control, via the left anterior regions, over threat compared to neutral words. Methodological differences between UHCs and rTMS, discussed previously, may account for the discrepancy between the current findings and those of d'Alfonso et al. (2000). Moreover, rTMS has been suggested to be unreliable at localising the DLPFC as the coil is often located more dorsally (i.e., over the premotor cortex) rather than on the DLPFC (Herwig et al. 2001). Therefore, the degree to which DLPFC activity had been modulated by rTMS is somewhat questionable. In contrast, the UHC method (Schiff and Lamon 1994) is assumed to increase DLPFC activity (Harmon-Jones 2006). However, it is important to note that the localisation of the UHC effects has yet to be explored with high spatial frequency methods.

The current results also conflict with the attentional bias literature that highlights a positive relationship between high anger and interference for threat (e.g. Smith and Waterman 2003; van Honk, et al. 2001). However, it is important to note that these studies did not explicitly explore attentional bias in relation to the effects of UHCs. An alternative view, based on the current study, is that during cognitive tasks RHCs affect frontal motivational and/or cognitive rather than anger-related networks. Indeed the facilitation effect observed only in the RHC condition is consistent with evidence that RHCs increase approach motivation (Harmon-Jones, 2006). See also Davidson and Irwin (1999) and Davidson, et al, (2000). As discussed in Chapter 1, according to Gray (1987) the approach based BAS motivates and guides behaviour towards rewarding goals. Therefore, threat-related facilitation found in the RHC condition may reflect increased activity of the BAS, which in turn promoted the avoidance of the threat to focus attention on goal relevant task responses.

The current findings are also consistent with view that left frontal brain activity reflects increased cognitive control over emotional responding (e.g. Davidson 2004). For example, the facilitated threat in the RHC condition may reflect increased cognitive control over goal directed behaviour in the presence of threat (Davidson 2004; Miller and Cohen 2001). That is, increased left frontal activity, via RHC, may have resulted in greater cognitive resources being applied to the task relevant processes. This interpretation is consistent with the view that the PFC maintains activity that represents goals, in this case colour responding, while simultaneously inhibiting or avoiding threat (Miller and Cohen 2001). It is also consistent with the view that, in ambiguous situations the PFC sends signals to other brain regions to facilitate task relevant responses even when potentially stronger alternatives, in this case threat information, compete for attention (Davidson 2003).

In summary, the current findings highlight the role of RHCs in the modulation of attentional bias to threat words. However, it is unclear whether it reflects approach-based motivational responses or cognitive control for increased goal attainment (see Miller and Cohen 2001; Davidson, 2003 for reviews). Furthermore, as only low-level behavioural data was gained in the current study no insight can be provided into underlying neuronal mechanisms related to the RHC effects on attentional bias to threat. By converging EST paradigms with ERP measures (see Chapter 2 for in depth discussion on ERP measures), it may be possible to gain insight into the specific underlying mechanisms of the facilitation effect found in the RHC condition. ERPs may also provide a means to begin to unravel whether the modified attentional bias in the RHC condition was a result of increased approach motivation or increased cognitive control.

Additionally, despite observing attentional bias in the RHC condition, it is not clear whether this effect may have been influenced by individual differences in trait anger. As individual differences in trait anger are associated with both greater left frontal brain activity (Harmon-Jones, 2003) and attentional bias to threat (e.g. van Honk, et al., 2001) it is important to explore how the effects of RHCs co-vary with individual differences in trait anger. These issues are explored in the next Chapter.

CHAPTER 4

STUDY 2: UHC MODULATION OF THREAT PROCESSING: AN ERP STUDY

4.1 Introduction

The aim of the study presented in this chapter was to explore the underlying processes of the induced facilitated threat responding found in the RHC condition in Study 1 (Chapter 3). More specifically it is important to understand whether this facilitation reflects changes in motivational or/and control processes. Given the results of Study 1 and the lack of research in this field, an investigation of attention related ERPs recorded during an EST paradigm used alongside the UHC method was warranted. As discussed in Chapter 3, the facilitated attentional bias found in Study 1 may have been confounded by individual differences in trait anger as this was not controlled for in Study 1. For this reason, Study 2 also aimed to explore how trait anger interacted with the effects of the UHC method during the attentional processing of threat-related words.

As discussed in Chapters 1 and 2, high trait AX-O, indexed by the STAXI-2 (Spielberger 1999), involves a tendency to engage in approach motivated aggression in the presence of angry feelings (e.g. see Carver and Harmon-Jones 2009 for a review). In contrast, low trait AX-O, involves greater withdrawal motivated tendencies whereby anger expression is suppressed (Carver and Harmon-Jones 2009). For this reason, high and low AX-O was used in the present Study to provide a means to explore how high anger influenced the effect of RHCs on threat-related attentional bias. From here on in, AX-O is simply referred to as anger.

As discussed in Chapter 1, attentional bias to threat is known to play an important role in automatic emotion regulation (e.g. Gross 1998). As such, gaining a better understanding of attentional bias involved in the expression of anger has practical implications for informing efficient anger reduction interventions in people susceptible to anger (Meier et al. 2006). Research has shown that high anger is related to attentional bias towards threat (interference) (e.g. Smith and Waterman 2003) an effect caused by deficits in later cognitive processes (e.g. van Honk et al. 2001; Anderson and Bushman 2002) possibly reflecting reduced ability to disengage from threat (Koster et al. 2004). This inability to disengage attention from threat is suggested to interfere with competing processes involved in goal related task performance (e.g. Stewart et al. 2010; Bishop et al. 2004). While this evidence allows an understanding of how later cognitive processes influence threat-related attentional bias and goal attainment, its correlational nature does not allow any examination of the direction of this relationship between anger and attentional bias to threat.

Exploring how the UHC method modulates underlying neural indices of attentional bias to threat may provide a better understanding of any causal relationship. As discussed in Chapter 1 and 2, the UHC literature has shown that RHCs increase left frontal brain activity, approach motivation (Harmon-Jones 2006) and aggression (Peterson et al. 2008). This evidence supports views that increased approach motivation guides and motivate goal relevant behaviour (for a review see, Harmon-Jones et al. 2010). In the case of anger, the goal relevant behaviour may be to approach threat in an attempt to remove the stressor. According to this view RHCs should increase approach motivated anger and subsequently increase attentional bias towards threat-related stimuli. This assumption is supported by evidence showing a positive association between left frontal activity and approach-motivated attentional bias towards threat (interference) (e.g. d'Alfonso et al. 2000; van Honk and

Schutter 2006). Findings from these studies suggest that approach motivated attentional bias may reflect a reduced ability to inhibit threat processing during competing tasks.

However, this assumption is inconsistent with the results of Study 1 where increased facilitation was found following RHCs (see Chapter 3) and also challenges the view that the left frontal brain plays a fundamental role in the cognitive control or inhibition of emotion based responding (e.g. Davidson 2004). According to this view, increased left frontal brain activity should reduce attentional bias towards threat and increase task relevant processing (approach motivation). Nevertheless, it remains unclear whether the increased threat-related facilitation effect found for RHCs in Study 1 reflected increased task relevant approach motivation or increased cognitive control over task relevant processing. As such further research exploring the underlying mechanism of the UHC effect in relation to trait anger was warranted. The focus of this study then will try to disentangle these two alternative explanations.

Furthermore, anger was not controlled for in Study 1 which might have accounted for some for the variance in the observed results. Therefore, it was decided then to replicate Study 1 but also to include the manipulation of anger as an additional independent variable. At this juncture it is important to note that according to the Diathesis Stress Model (Davidson 1992; Davidson and Tomarken 1989), RHCs would not be sufficient to induce a change in affective behaviour. Instead modulations of attentional bias to threat would only be expected in response to RHCs if an individual was predisposed to high anger.

It follows then that if RHCs increases anger/approach motivation then any effects found for RHCs would be expected to be summative, in that they would be larger in individuals with

high compared to low trait anger. Conversely if RHCs increase cognitive control then any effects of high trait anger on attentional bias should be reduced.

As discussed in Chapters 1 and 2, ERPs provide a sensitive measure to explore the underlying processes of the facilitation effect found in Study 1. However research exploring ERPs related to anger-related attentional bias is rare (e.g. Stewart et al. 2010; Bertsch et al. 2009). Evidence does suggest that such attentional bias is confined to later cognitive ERP components, namely the P3b, N2 and NSW (Stewart et al. 2010). For this reason the present study focused separately on UHC related modulations of task related approach motivation (indexed by P3b amplitude) and cognitive control (indexed by N2 and NSW) and during the EST. It is important to note that to date no research has yet to explore UHC effects on attention related ERPs and as such the present study is exploratory in nature.

The centroparietal P3b indexes attentional resource allocation towards both top-down and bottom-up motivationally relevant information (e.g. MacNamara et al. 2009). It is therefore appropriate in the present study for investigating the effects of UHCs and trait anger on approach/task motivation related attentional processing. Evidence indicates that P3b is larger for threat than neutral words in high anger (Stewart et al. 2010). In accordance with previous evidence (e.g. Kissler, Herbert, Winkler, and Junghöfer 2009; Cuthbert, Schupp, Bradley, Birbaumer, and Lang 2000) this finding suggests that high anger participants found threat words more emotionally relevant. Evidence also suggests that smaller P3b for threat compared to neutral targets denotes increased task difficulty and shows threat interfered with target responding (Duncan-Johnson and Donchin, 1977; MacNamara et al. 2009).

The frontocentral N2 is thought to reflect the extent that attentional control is recruited to resolve conflict and inhibit incorrect responses (Braver et al. 2001; Jones, Cho, Nystrom, Cohen, and Braver 2002; Nieuwenhuis et al. 2003; van Veen and Carter 2002) and a "gating" mechanism where motivationally relevant information gains access to cognitive control systems (Dennis and Chen 2007; Dennis 2009). Therefore, this component provides a neural basis for emotion-cognition integration and is appropriate to examine the effects of UHC and trait anger on attentional control processing. Evidence indicates that N2 is larger for threat than neutral stimuli in high anger participants (Stewart et al. 2010). According to Neural Efficiency (e.g. Dennis and Chen 2007; Gray 2004) and Dual System Models (Botvinick et al. 2001), this finding suggests that high anger participants use more inhibitory resources to suppress conflict and monitor actions during the presence of threat.

The frontocentral NSW provides a neural index for conceptual level inhibition and efficiency during EST conflict, (West 2003; West and Alain 2000) with larger NSW for threat compared to neutral words denoting threat-related interference (van Hooff, et al. 2008). Previous research has shown larger NSW amplitudes for threat than neutral words in high anger participants (Stewart et al. 2010). These results suggest that high anger participants will experience increased threat-related interference reflecting reduced efficiency of cognitive control mechanisms. The NSW therefore provides a useful index of the extent that cognitive control is implemented during task relevant processing as well as an index of the efficiency of inhibitory mechanisms used to supress threat conflict in the EST in the current study. Despite a relative paucity of literature in this area predictions have been made regarding EST RTs and ERP amplitude in response to threat versus neutral words relative to UHC and anger.

4.1.1 Predictions

It is unclear at present whether trait anger influenced the facilitation effect of the RHC in Study 1, so predictions were guided by 1) previous evidence that shows RHCs increase approach motivation and aggression (Harmon-Jones 2006; Peterson et al. 2008), 2) Interference for threat is found in high anger participants (e.g. Smith and Waterman 2003) and 3) Theories that suggest that RHCs would only induce affective behavioural changes in participants predisposed to high anger (e.g. Davidson 1992).

4.1.1.1 Behavioural element

Given that both high trait anger (e.g. Smith and Waterman 2003) and greater left frontal brain activity (e.g. d'Alfonso et al. 2000) is related to increased EST interference, the following predictions were made about the effects of RHCs on attentional bias in the EST.

It was hypothesised that if the RHCs facilitation effects found in Study 1, was related to increase approach motivated anger then:

1) There would be no main effects of UHC, anger or word-type on EST bias-scores
2) Attentional bias towards threat compared to neutral words would be greater in RHC compared to LHC groups.
3) Attentional bias towards threat compared to neutral words would be greater in high compared to low anger participants.
4) Compared to LHCs, RHCs would increase interference to threat compared to neutral words and this effect would be greater in high than low anger participants.

4.1.1.2 ERP element

ERP predictions were based on evidence that high compared to low trait anger is evidenced by enhanced P3b (increased approach motivation), N2 and NSW amplitudes (increased cognitive control/ reduced inhibitory efficiency) for threat compared to neutral words in the EST (Stewart et al. 2010). The exploration of these ERP components addresses the possibility that the facilitation observed in Study 1 could either be explained by motivation or/and control. Predictions regarding laterality were made on the hypothesised effect of UHCs on contralateral brain activity.

It was hypothesised that if RHCs increase approach motivated anger that:

5) No amplitude main effects would be observed at P3b, N2, or NSW in either the anger, word-type or hemisphere conditions.

6) Relative to LHCs, RHCs would increase P3b, N2, and NSW amplitudes for threat compared to neutral words.

7) The P3b, N2, and NSW increased amplitude difference between threat and neutral words would be greater in high compared to low anger participants.

8) Relative to LHCs, RHCs would increase P3b, N2, and NSW amplitudes for threat compared to neutral words and this effect would be larger in high than low anger participants.

9) P3b, N2, and NSW amplitudes would be larger in the left than the right hemisphere for RHCs compared to LHCs.

10) P3b, N2, and NSW amplitudes would be larger in the left than the right hemisphere for high compared to low anger participants.

11) P3b, N2, and NSW amplitudes would be larger in the left than the right hemisphere for RHCs compared to LHCs and this effect will be larger in high compared to low anger participants.

4.2 Method

4.2.1 Design

4.2.1.1 Behavioural element

The behavioural element of the study used a 2 between subject (UHC group; RHC, LHC) × 2 between subject (Anger; high, low) × 2 within subject (word-type; threat, neutral) mixed factorial design to examine the effects of UHC on attentional bias to threat. UHC and anger expression were between subject factors and word-type was a repeated measures factor. The dependent variable was EST RTs (ms).

4.2.1.2 ERP element

To examine the effects of the UHC method on motivational aspects of attentional bias to threat the study employed a 2 between subject (UHC group; RHC, LHC) × 2 between subject (Anger; high, low) × 2 within subject (word-type; threat, neutral) × 3 within subject (Region, frontal, frontocentral, central) × 2 within subject (laterality, left, right) mixed factorial design. The dependent variable was P3b amplitude.

To examine the effects of UHC on neural correlates of cognitive control of attentional bias to threat the study employed a 2 between subject (UHC group; RHC, LHC) × 2 between subject (Anger; high, low) × 2 within subject (word-type; threat, neutral) × 3 within subject (Region,

frontal, frontocentral, central) × 2 within subject (laterality, left, right) mixed factorial design. The dependent variables were N2 and NSW amplitude.

4.2.2 Participants

Participants were 38 undergraduates who scored in the top or bottom 20% of the AX-O STAXI-2 subscale (Spielberger 1999) during the sampling phase of the study (see section 2.2) Participants also met the inclusion criteria as described in section 2.2.

Participants were allocated to the RHC or LHC group in a Latin square order, based on whether they scored high or low in AX-O. High anger participants (M = 20.67, SD = 3.45) scored significantly higher in the AX-O subscale than low anger (M = 12.06, SD = 1.25) participants, $F(1, 36) = 95.06$, $p < .001$. The RHC group [11 participants were classified as high anger (AX-O score M = 20.73, SE = 1.05) and 8 were classified as low anger (AX-O score M = 12.25, SE = .45)] were five males (aged; M = 20.60, SD = 1.4) and 14 females (aged; M = 20.07, SD = .62). In total the mean anger expression-out score of the RHC group was, M =17.16, SD = 5.08. The LHC group [10 participants were classified as high anger (AX-O score M = 20.60, SE = 1.15) and 9 were classified as low anger (AX-O score M = 11.89, SE = .42)] were three males (aged; M = 23.00, SD = 2.52) and 16 females (aged; M = 21.43, SD = 1.48). In total the mean anger expression out score of the LHC group was, M=16.47, SD = 5.22. Analysis revealed that the RHC and the LHC group did not differ in relation to trait anger, $F(1, 36) = .17$, $p > .05$. Participants received course credits for their participation. Coventry University Ethics Committee approved the investigation and written informed consent was gained from each participant.

4.2.3 Materials

4.2.3.1 Word stimuli

Information about the generation of the EST words can be found in Section 2.5.1.2. The words used in this Study are also presented in Appendix 1.

4.2.3.2 The Emotional Stroop Task (EST)

.Information about the EST protocol is presented in Section 2.5.1.1.

4.2.4 The UHC method

Information about the UHC procedure is presented in detail in Section 2.4.

4.2.5 Procedure

Before the EST, participants were given a participant information sheet, provided informed consent and were prepared for EEG recordings. Participants completed the EST in a single session and UHC group allocation was subject to Latin squared counterbalancing according to AX-O scores. Participants were prepared for EEG (see Section 2.7.1 for information about this procedure) and standardised verbal instructions on how to complete the designated hand contraction were provided. During the EST, participants were seated 127cm from a computer monitor and responded as quickly and accurately as possible to the font colour presented, through a button press. The experiment lasted 40 minutes, with 5-minute breaks between blocks to minimise fatigue effects.

4.2.6 EEG Acquisition

Information regarding data acquisition procedures is presented in Section 2.7.2.

4.2.7 Data Reduction and Statistical Analysis

4.2.7.1 Preparation of Behavioural Data

All incorrect or missed responses were rejected as error. The percentage of data rejected as an error was *M* = 1.85%, *SD* = 2.51. Response times < 300ms or >1500ms were also removed. In this instance, the percentage of rejected data was *M*= 1.44%, *SD* = 1.80. RTs less or more than three standard deviations above each participant's mean (*M*= .87%, *SD* = 1.8) were also removed as outliers (cf. Putman, Arias-Garcia, Pantazi, van Schie 2012). The final analysis consisted of 38 datasets. An alpha level of 0.05 was applied for all statistical tests unless stated otherwise and all analyses were performed two-tailed.

4.2.7.2 Preparation of EEG Data

Continuous EEG raw data was processed according to the procedure set out in Section 2.8. As discussed in Section 2.8, separate grand average ERPS were computed for each condition; RHC threat words, RHC neutral words LHC threat words and LHC neutral words. Electrode positions and scoring epochs P3b (340-580ms), N2 (137-223ms), and NSW (330-700ms) were selected following visual inspections of grand ERP waveforms and referring to previous EST ERP literature (e.g. Thomas et al. 2007; Stewart et al. 2010; van Hoof et al. 2008). The P3b amplitude was recorded at centroparietal (CP1 and CP2), parietal (P3 and P4), and parietooccipital (PO3, and PO4) sites. The N2 amplitude was recorded at frontal (F3 and F4), frontocentral (FC1 and FC2), and central (C3and C4) electrode sites. The NSW amplitude was recorded at frontal (F3 F4), frontocentral (FC1 and FC2), and central (C3 and C4) sites.

4.3 Results

For brevity sake only significant inferential statistics that directly correspond to the predictions (Section 4.1.1) are presented in detail. All other results are presented in table format.

4.3.1 Behavioural Element

To explore if UHCs modulated RTs in relation to trait anger, a 2 (UHC group; RHC, LHC) × 2 (Anger; high, low) × 2 (word-type; threat, neutral) mixed analysis of variance (ANOVA) was performed. Bonferroni corrections were applied for multiple comparisons. Mean RTs and standard deviations for threat and neutral words in the RHC and LHC groups in relation to high and low anger are shown in Table 4.1.

Table 4.1. Mean EST RTs and standard deviations in parenthesis for threat and neutral words in the RHC and the LHC groups in relation to anger

UHC group	**Anger level**	***(N)***	**Word-type**	***M* (*SD*)**
RHC	High	*(10)*	Threat	776.17 (94.73)
			Neutral	768.55 (99.40)
	Low	*(8)*	Threat	738.25 (124.95)
			Neutral	749.16 (124.73)
LHC	High	*(10)*	Threat	802.41(116.69)
			Neutral	808.21 (113.20)
	Low	*(9)*	Threat	825.88 (152.89)
			Neutral	812.19 (150.50)

Kolmogorov-Smirnov indicated that the threat, D (19) = 0.14, p > .05, and neutral condition, D (19) = 0.15, p > .05, in the RHC group and the threat, D (19) = 0.13, p > .05, and neutral condition, D (1y9) = 0.13, p > .05, in the LHC group, were normally distributed. This test also indicated that threat, D (21) = 0.12, p > .05, and neutral condition, D (21) = 0.15, p > .05, in the high anger group and the threat, D (17) = 0.14, p > .05, and neutral condition, D (17) = 0.17, p > .05, in the low anger group, were normally distributed. Levene's tests showed that the variance between RTs in the UHC and anger group were equal for threat, F (1, 36) = .17, p = .77, and neutral conditions, F (1, 36) = .024, p = .87.

As anticipated in prediction 1, the ANOVA revealed no significant RT differences between threat and neutral words, F (1, 33) = .15, p >.05, ηp^2 = .00, RHCs and LHCs group, F (1, 33) = 1.80, p >.05, np^2 = .05, or high and low anger, F (1, 33) = .03, p >.05, np^2 = .00. Contrary to predictions 2 and 3, analysis showed no RT word × hand interaction, F (1, 33) = 87, p > .05, np^2 = .03 or word × anger interaction, F (1, 33) = 01, p > .05, np^2 = .00.

In partial support of prediction 4, analysis revealed a significant hand × anger × word interaction, F (1, 33) = 10.03, p < .005, np^2 = .23. However, contrary to predictions post hoc tests revealed no significant word-type RT difference between the UHC groups in high anger participants, F (1, 19) = 1.75, p > .05, np^2 = .09. However, in low anger participants, RTs for threat compared neutral words were significantly different in the RHC compared to the LHC group, F (1, 15) = 18.85, p < .05, np^2 = .56. Further analysis revealed that there was no RT difference for threat and neutral words in the RHC group t (7) = -2.20, p > .05. Surprisingly in the LHC group RTs were significantly slower for threat than neutral words, t (7) = 4.51, p < .05. This indicates that in high anger participants UHCs did not influence attentional bias

to threat but that in low anger participants, LHC's and not RHC's increased threat-related interference. These findings are presented in Figure 4.1.

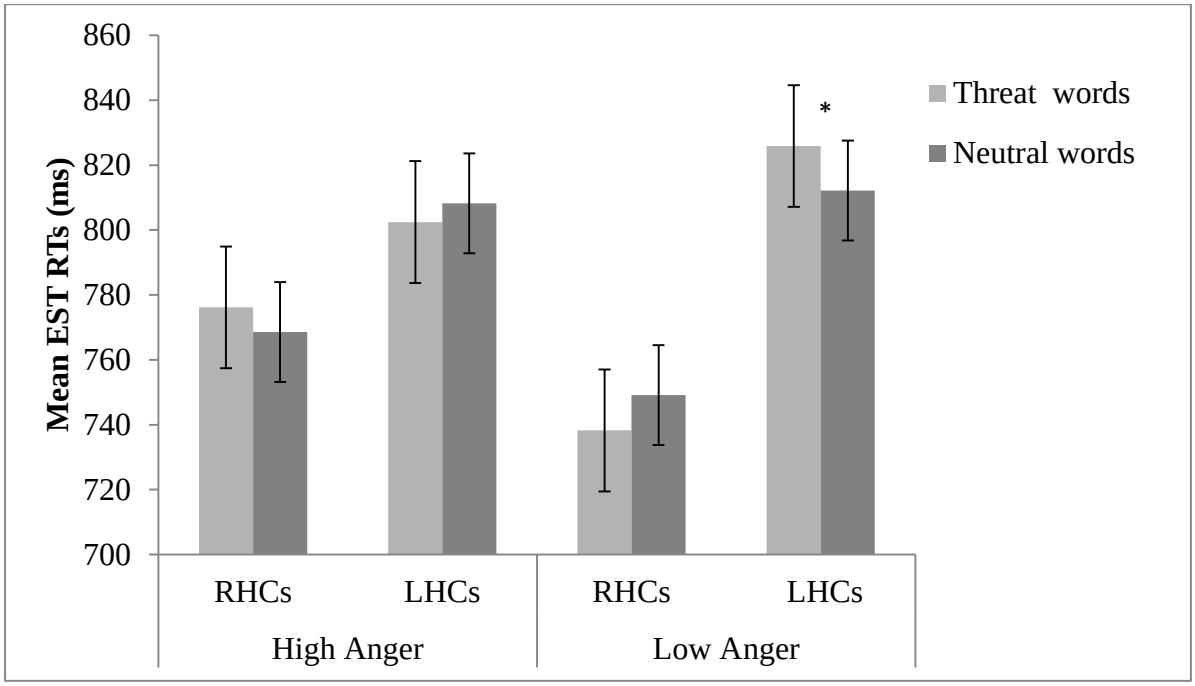


Figure 4.1. Mean EST RTs (ms) and S.E.M for threat and neutral words in the RHC compared to the LHC group for high and low anger participants. Larger RTs to threat than neutral words indicate threat-related interference and smaller RT to threat than neutral words indicate threat avoidance and EST facilitation

4.3.2 ERP Element

For interpretational ease, P3b, N2, and NSW amplitude data were individually subjected to a series of separate 5-way mixed ANOVAs that compared main effects and interactions that occurred in response to UHC, anger, word-type, region and hemisphere. Significant interactions that included only within subject factors were explored using repeated measure contrasts and interactions that included both within and between subject factors were explored using one-way ANOVA and paired t-tests. Bonferonni corrections were applied for multiple comparisons. As discussed in depth in Section 2.9.1, type 1 errors were further protected by applying Greenhouse-Geisser epsilon adjustment for all ERP results presented in this Chapter.

4.3.2.1 P3b amplitude

To explore if RHCs modulated motivational indices of attentional bias to threat in relation to anger, a 2 (UHC group; RHC, LHC) × 2 (anger; high, low) × 2 (Word-type; threat, neutral) × 2 (Hemisphere; left, right) × 3 (Region; centroparietal, parietal, parietooccipital) mixed ANOVA was performed on P3b mean amplitudes. Descriptive statistics of P3b are presented in Table 4.2. Inferential statistics of this analysis are presented in Table 4.3.

Table 4.2. P3b mean amplitudes (μV) at left and right, centroparietal, parietal and parietooccipital sites for threat and neutral words in the UHC conditions for high and low anger participants. Standard deviations are in parenthesis.

		RHC		LHC	
		High anger (N = 10)	**Low anger (N = 8)**	**High anger (N = 10)**	**Low anger (N = 9)**
Threat Words	**CP1**	-.97 (3.20)	-.33 (3.59)	-.13 (1.95)	-.80 (1.63)
	CP2	-.45 (3.34)	.22 (4.16)	.47 (1.50)	.09 (1.63)
	P3	2.46 (2.72)	1.46 (4.67)	1.24 (1.56)	1.02 (.91)
	P4	2.35 (3.27)	1.20 (2.77)	1.72 (1.96)	2.39 (1.38)
	PO3	5.32 (4.61)	3.60 (5.52)	2.48 (3.02)	2.73 (1.85)
	PO4	3.66 (2.54)	3.31 (5.19)	2.81 (3.35)	2.93 (2.88)
Neutral Words	**CP1**	-.23 (2.77)	-.69 (3.41)	-.45 (2.30)	-.77 (1.93)
	CP2	.20 (2.76)	.01 (4.38)	-.06 (1.37)	.23 (1.89)
	P3	2.89 (2.13)	1.52 (4.85)	1.73 (1.45)	.90 (1.15)
	P4	2.94 (2.67)	1.47 (2.76)	1.80 (1.57)	1.83 (1.68)
	PO3	5.47 (4.76)	3.88 (5.55)	3.45 (2.89)	3.74 (2.34)
	PO4	3.93 (2.26)	4.03 (4.80)	2.88 (2.93)	2.59 (3.33)

Table 4.3. Inferential statistics for effects of UHC, anger, word, hemisphere and region on P3b mean amplitudes (μV). Significant effects are highlighted.

Condition	***F***	***p***
Anger	.34	.57
Hand	.65	.43
Word	2.58	.12
Word x UHC	.89	.35
Word x Anger	.89	.35
Word x UHC x Anger	.28	.60
Region	25.45	.000
Region x UHC	.76	.41
Region x Anger	.17	.71
Region x UHC x Anger	.34	.59
Hemisphere	.14	.72
Hemisphere x UHC	.56	.46
Hemisphere x Anger	.38	.54
Hemisphere x UHC x Anger	.02	.89
Word x Region	2.03	.15
Word x Region x UHC	.94	.39
Word x Region x Anger	1.04	.36
Word x region x UHC x Anger	4.16	.03
Word x Hemisphere	2.20	.15
Word x Hemisphere x UHC	8.16	.01
Word x Hemisphere x Anger	.08	.78
Word x Hemisphere x UHC x Anger	.31	.58
Region x Hemisphere	3.70	.04
Region x Hemisphere x UHC	.25	.74
Region x Hemisphere x Anger	.06	.92
Region x Hemisphere x UHC x Anger	1.36	.26
Word x Region x Hemisphere	1.90	.16
Word x Region x Hemisphere x UHC	4.75	.01
Word x Region x Hemisphere x Anger	.34	.70
Word x Region x Hemisphere x UHC x Anger	.55	.57

In support of prediction 5, no main effect of UHC, anger, word-type or hemisphere was found (p >.05). These findings are presented in Table 4.3. However, contrary to prediction 5, the ANOVA revealed a main effect of region, F (1.13, 37.13) = 25.45, p <.001, np^2 = .44 with significantly greater P3b at parietooccipital than parietal, F (1, 33) = 24.25, p <.001, np^2 =.42 and centroparietal sites, F (1, 33) = 27.15, p <.001, np^2 =.45. This can be seen in Figures 4.2.

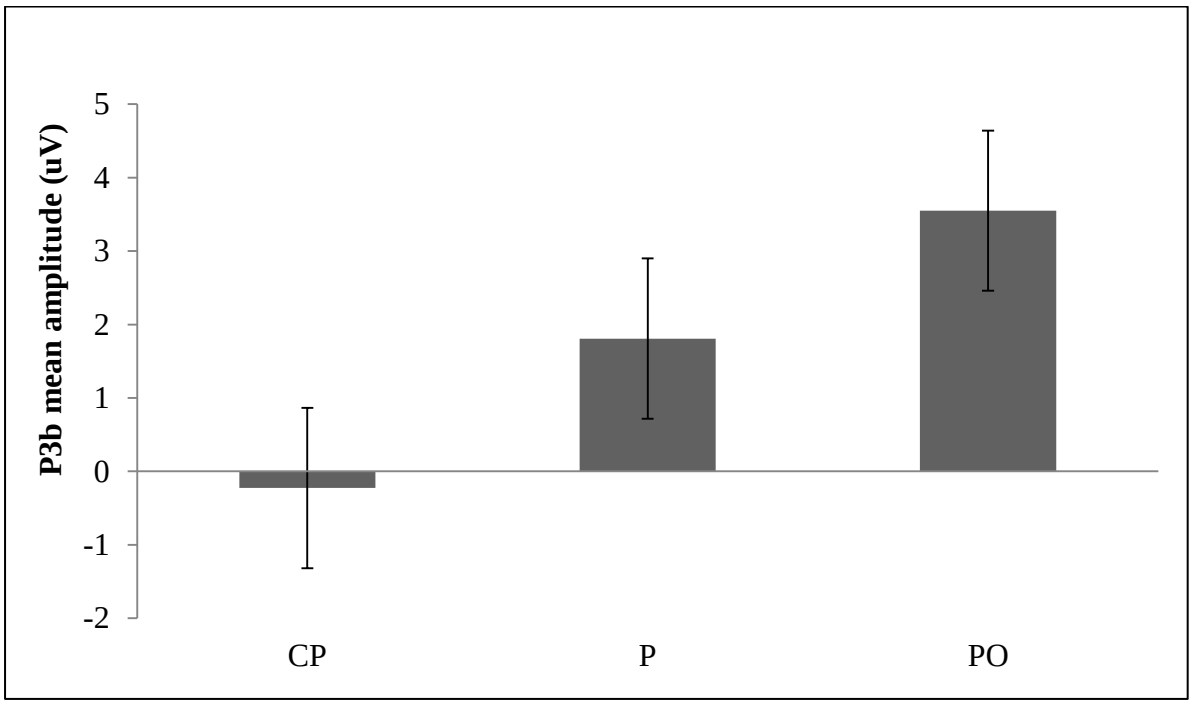


Figure 4.2. P3b mean amplitudes (μV) at centroparietal (CP), parietal (P) and parietooccipital (PO) sites. Data are expressed as mean (SEM).

With respect to prediction 6, that RHCs compared to LHCs would increase P3b for threat compared to neutral words, no UHC × word interaction was found (p >.05, see Table 4.5). However, the ANOVA revealed a significant UHC × word × hemisphere interaction, F (1.00, 33.00) = 8.16, p <.05, np^2 =.20. This interaction was qualified by a significant UHC × word × region × hemisphere interaction, F (1.90, 62.84) = 4.75, p <.05, $np2$ =13. However, contrary to predictions, post hoc tests revealed that in the RHC group no significant P3b word-type difference was observed at any region in either hemisphere (p >.05). In the LHC condition, no significant P3b word-type difference was observed at centroparietal and parietal regions in either hemisphere (p >.05). However, at left parietooccipital sites, LHCs significantly reduced P3b for threat compared to neutral words, t (18) = -2.80, p < .05, but following Bonferroni corrections these differences failed to reach significance. Nevertheless, this pattern can be seen in Figures 4.3 and 4.4.

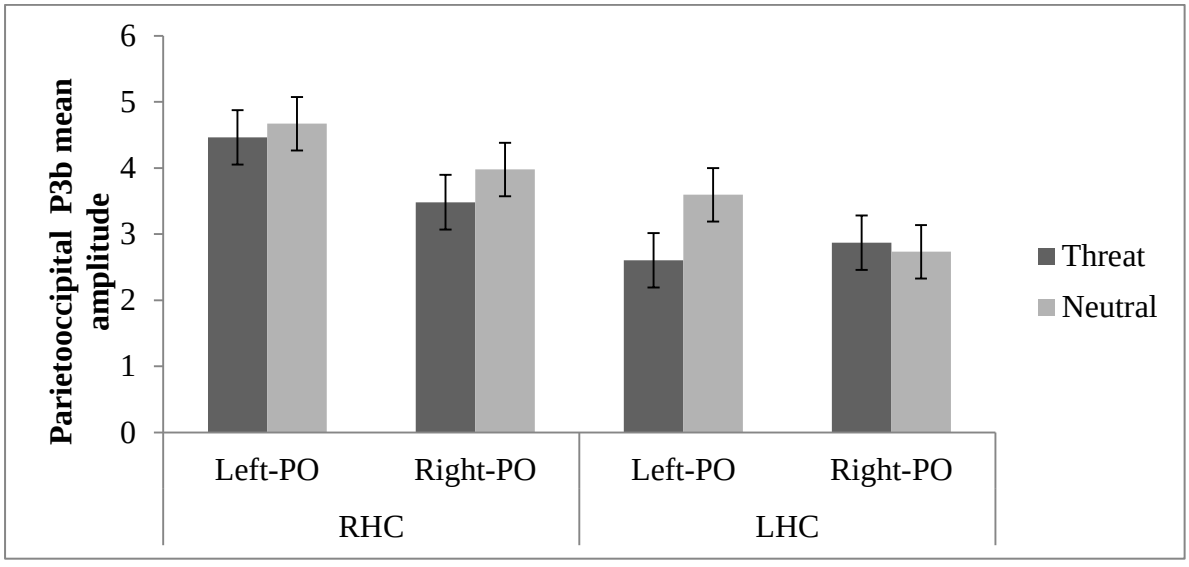


Figure 4.3. P3b mean amplitudes (μV) at left and right parietooccipital (PO) sites for threat and neutral words in the RHC and LHC group. Data are expressed as mean (SEM).

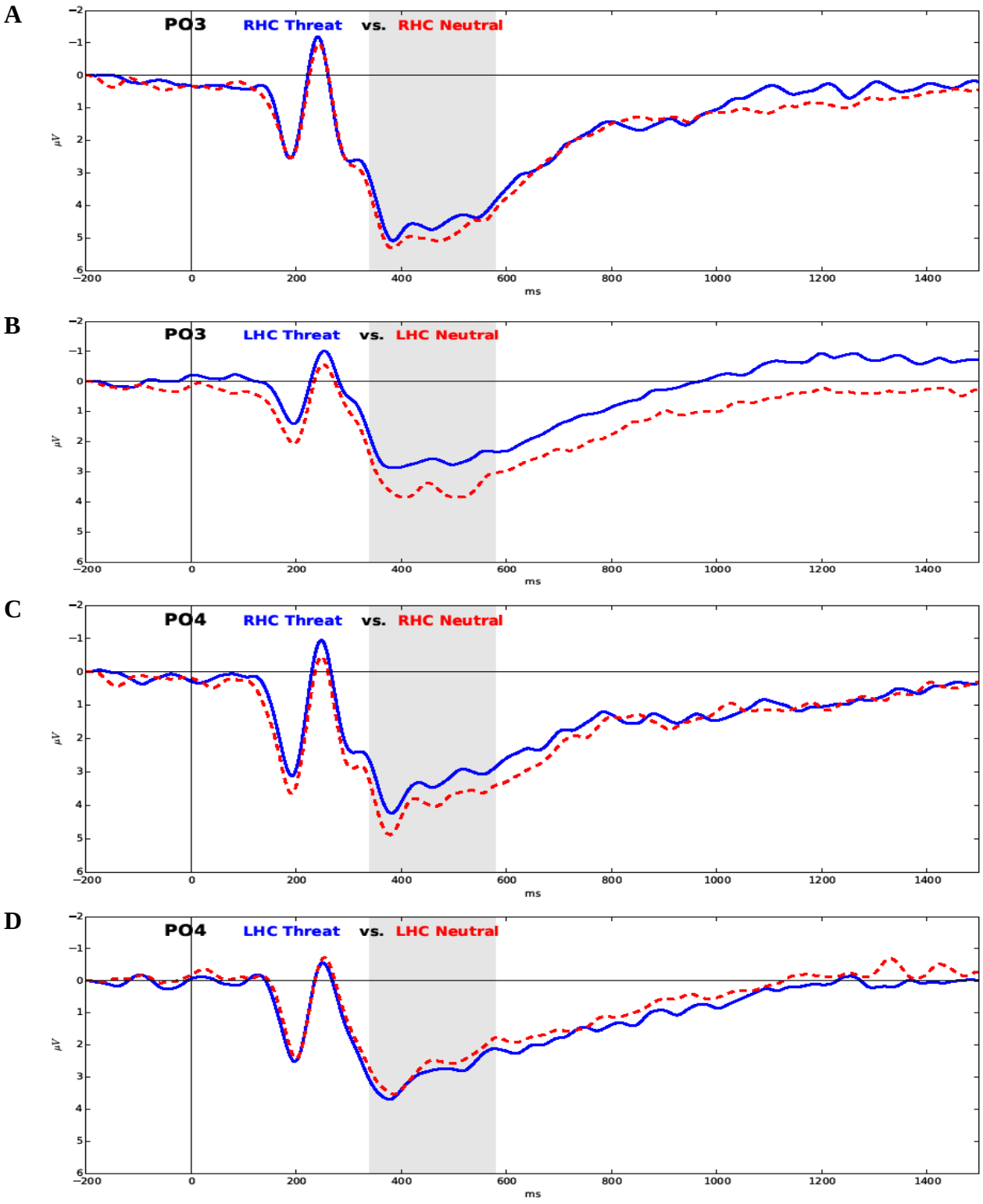


Figure 4.4. Grand average waveforms of P3b amplitudes (μV) (shaded grey) at left and right parietooccipital sites (PO3 and PO4 respectively) for threat and neutral words in the RHC (Panels A and C) and LHC group (Panels B and D)

With respect to prediction 7, no word × anger P3b amplitude interaction was found, (p > 0.05, see Table 4.5). Furthermore, with regards to prediction 8, the ANOVA revealed no UHC × anger × word interaction (p > 0.05, see Table 4.5). However, a significant UHC × anger × word × region interaction was observed, F (1.62, 53.50) = 4.16, p <.05, np^2 =.11. Although contrary to predictions, post hoc tests revealed that in the RHC group, no P3b amplitude word-type difference was observed at any regional site in high anger participants or at parietal or parietooccipital sites, in low anger participants (p >.05). However, at centroparietal sites, low anger participants in the RHC group showed greater P3b amplitude for threat compared to neutral words, t (7) = 2.40, p <.05, but again after Bonferroni corrections this difference was not significant. In the LHC group no P3b amplitude word-type difference was observed at any regional site in low anger participants (p >.05) or at parietal or parietooccipital sites in high anger participants, (p >.05) but at centroparietal sites, P3b was significantly larger for threat than neutral words, t (9) = 2.66, p < .05. Though after Bonferroni corrections, this difference failed to reach significance. These patterns of centroparietal P3b amplitude word-type differences for UHCs in relation to trait anger can be seen in Figures 4.5, 4.6 and 4.7.

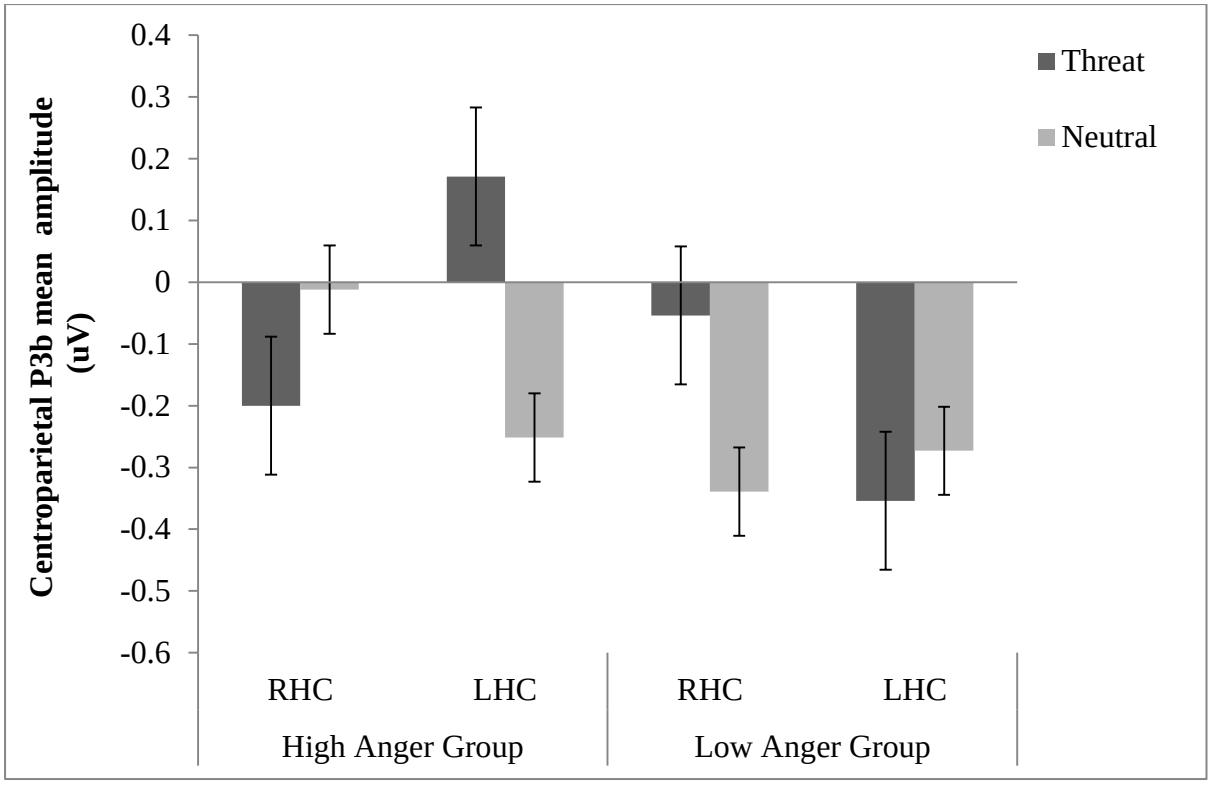


Figure 4.5. Centroparietal P3b mean amplitude (μV) for threat and neutral words for the RHC and LHC groups in high and low trait anger participants. Data are expressed as mean (SEM)

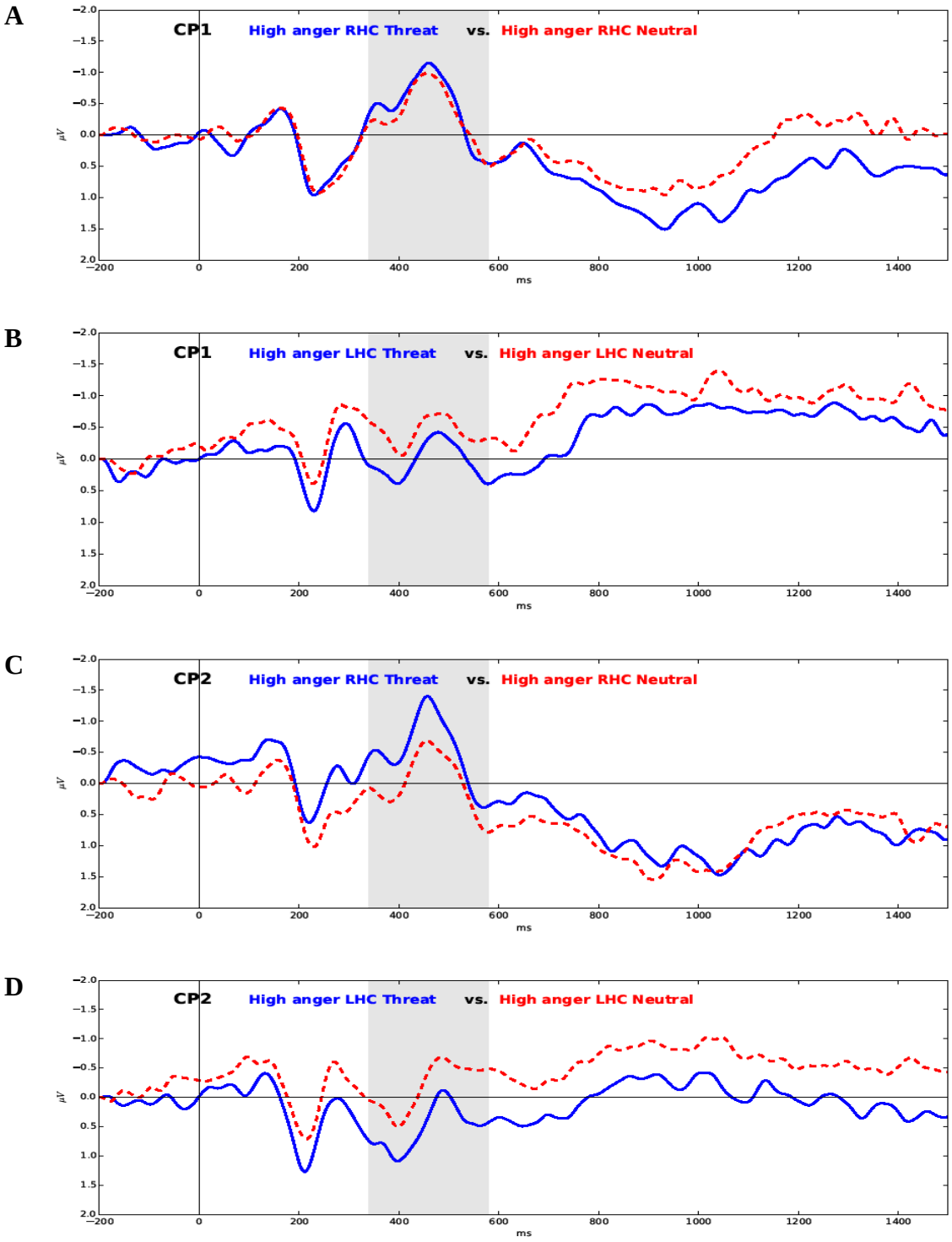


Figure 4.6. Grand average waveform showing centroparietal P3b amplitudes (μV), (shaded grey), for threat and neutral words in high anger participants in the RHC and LHC groups. Illustrates that high anger participants exhibited larger centroparietal P3b for threat than neutral words following LHCs (Panel B and D) compared to RHCs (Panels A and C)

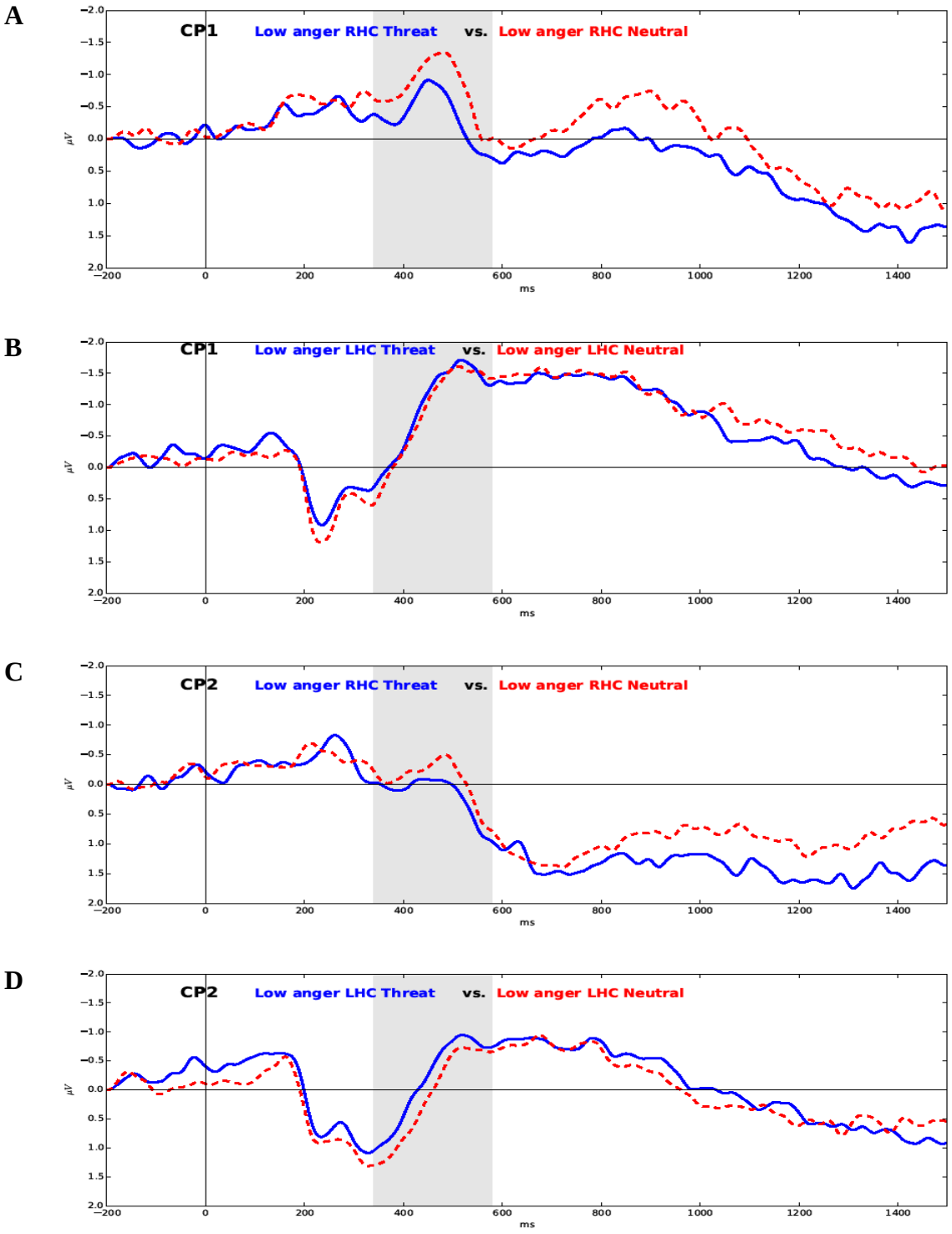


Figure 4.7. Grand average waveforms showing centroparietal P3b amplitudes (μV) (shaded grey) for threat and neutral words in low anger participants in the RHC and LHC groups. This illustrates that low anger participants exhibited larger centroparietal P3b for threat than neutral words following RHCs (Panel A and C) compared to LHCs (Panels B and D)

Contrary to predictions 9 and 10, no UHC × hemisphere interaction and no anger × hemisphere interaction were found ($p > 0.05$). Contrary to prediction 11, no UHC × anger × hemisphere interaction observed ($p > 0.05$). These findings are presented in Table 4.5.

4.3.2.2 N2 amplitude

To explore if RHCs modulated cognitive control indices of attentional bias to threat in relation to anger, a 2 (UHC group; RHC, LHC) ×2 (Trait anger; high, low) x 2 (Word-type; threat, neutral) × 2 (Hemisphere; left, right) × 3 (Region; frontal, frontocentral, central) mixed ANOVA was performed on N2 peak amplitudes. Descriptive statistics for N2 amplitudes are presented in Table 4.4 and all inferential statistics of this analysis are presented in Table 4.5.

Table 4.4. The N2 peak amplitudes (μV) at left and right, frontal, frontocentral and central sites for threat and neutral words in the RHC and LHC conditions for high and low anger participants. Standard deviations are in parenthesis.

		RHC		LHC	
		High anger (N = 10)	**Low anger (N = 8)**	**High anger (N = 10)**	**Low anger (N = 9)**
Threat Words	**F3**	-.43 (.58)	.00 (.82)	-.22 (.56)	-.60 (.93)
	F4	-.59 (.64)	-1.15 (1.77)	-.31 (1.13)	-1.09 (1.06)
	FC1	-.97 (.85)	-1.13 (1.68)	.23 (1.13)	-.81 (.79)
	FC2	-.73 (.93)	-1.06 (1.61)	-.11 (.84)	-1.30 (1.07)
	C3	-.47 (.79)	-.65 (1.05)	-.15 (1.12)	.02 (.89)
	C4	-.53 (.90)	-.81 (1.46)	-.02 (1.00)	-.50 (.72)
Neutral Words	**F3**	-.68 (80)	-.40 (.94)	-.20 (.73)	-.08 (.93)
	F4	-.82 (.86)	-1.03 (1.21)	-.02 (.65)	-1.00 (1.29)
	FC1	-.83 (1.17)	-1.31 (2.12)	-.29 (1.25)	-.68 (1.02)
	FC2	-.81 (.91)	-.99 (2.17)	-.25 (.85)	-1.16 (1.15)
	C3	-.57 (1.19)	-.72 (1.25)	-.40 (1.33)	-.09 (.70)
	C4	-.73 (1.00)	-.81 (1.75)	.04 (.90)	-.74 (.50)

Table 4.5. Inferential statistics for effects of UHC, anger, word, hemisphere and region on N2 peak amplitude (μV). Significant effects are highlighted.

Condition	***F***	***p***
Anger	1.40	.25
Hand	1.50	.23
Word	.47	.50
Word x UHC	.46	.51
Word x Anger	6.20	.44
Word x UHC x Anger	.20	.66
Region	4.83	.01
Region x UHC	.83	.44
Region x Anger	2.05	.14
Region x UHC x Anger	1.05	.35
Hemisphere	4.87	.03
Hemisphere x UHC	.32	.58
Hemisphere x Anger	5.20	.03
Hemisphere x UHC x Anger	1.16	.29
Word x Region	.84	.43
Word x Region x UHC	3.78	.03
Word x Region x Anger	.35	.70
Word x region x UHC x Anger	1.73	.19
Word x Hemisphere	.50	.49
Word x Hemisphere x UHC	.01	.92
Word x Hemisphere x Anger	.07	.80
Word x Hemisphere x UHC x Anger	3.64	.07
Region x Hemisphere	2.41	.10
Region x Hemisphere x UHC	1.99	.15
Region x Hemisphere x Anger	1.60	.21
Region x Hemisphere x UHC x Anger	.80	.45
Word x Region x Hemisphere	.09	.92
Word x Region x Hemisphere x UHC	1.41	.25
Word x Region x Hemisphere x Anger	.15	.86
Word x Region x Hemisphere x UHC x Anger	.42	.66

Contrary to prediction 5, the ANOVA revealed a significant main effect of hemisphere, with smaller N2 negativity in the left than the right hemisphere, F (1.00, 33.00) = 4.87, p <.05, np^2 =.13. The ANOVA also revealed a significant main effect of region**,** F (1.85, 61.20) = 4.83, p <.05, np^2 =.13 with larger N2 at frontocentral than central sites, F (1, 33) = 12.60, p <.05, np^2 =.28 and a trend for larger N2 at frontocentral than frontal sites, F (1, 33) = .38, p =.06, np^2 =.10. This can be seen in Figure 4.8.

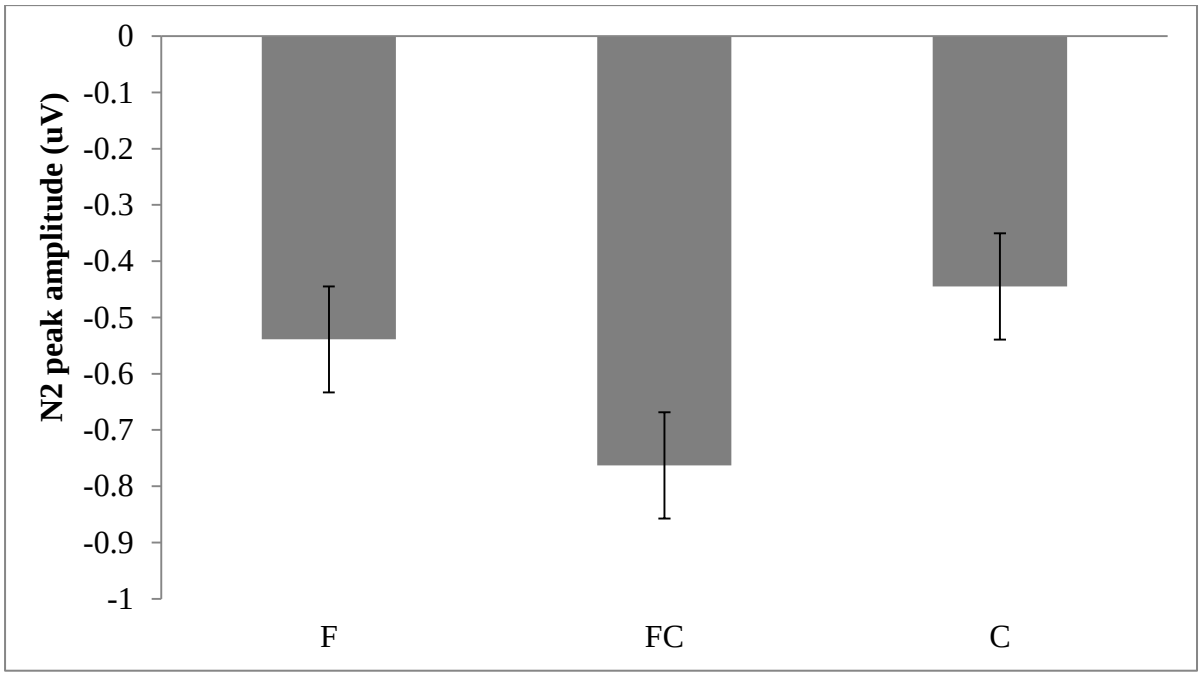


Figure 4.8. N2 peak amplitudes (μV) at frontal (F), frontocentral (FC) and central (C) electrode sites. Data are expressed as mean (SEM)

With respect to prediction 6, the ANOVA revealed no significant UHC × word interaction, (p >.05) see Table 4.3. However, a significant UHC × word × region interaction was observed, F (1.94, 64.04) = 3.78, p <.05, np^2 =.10. Post hoc tests revealed that the RHC group showed no N2 amplitude word-type difference at any regional sites (p >.05). The LHC group showed no N2 amplitude word-type difference at central or frontocentral sites (p >.05) but a trend emerged for larger N2 amplitudes for threat compared to neutral words at frontal sites, t (18) = -1.91, p = .07. However, following Bonferroni adjustments this difference failed to reach significance.

Predictions 7 and 8 were also unsupported; as the ANOVA showed no anger × word-type P3b amplitude interaction and no UHC × anger × word P3b amplitude interaction, (p >.05, see Table 4.3). Furthermore, contrary to predictions 9, no UHC × hemisphere interaction was observed (p >.05, see Table 4.3). However, in partial support of predictions 10, the ANOVA revealed a significant anger × hemisphere interaction, F (1.00, 33.00) = 5.20, p <.05, np^2 =.14. Though contrary to predictions, post-hoc tests revealed, no N2 amplitude laterality difference in high anger participants, (p > .05) but in low anger participants, N2 amplitude was significantly larger in the right than the left hemisphere, t (16) = 3.29, p < .01. This indicated that low anger participants recruited greater cognitive control in the right than left hemisphere regardless of UHC and word-type. This can be seen in Figure 4.9. Prediction 11 was unsupported as the ANOVA showed no UHC × anger × hemisphere P3b amplitude interaction (p >.05, see Table 4.3).

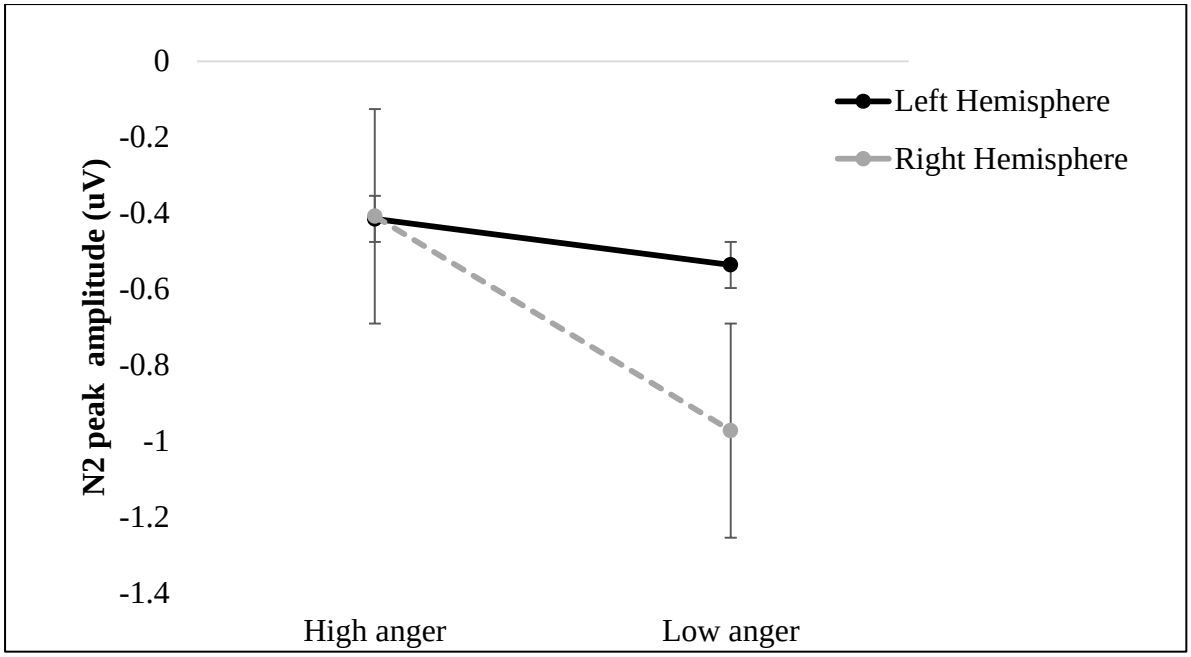


Figure 4.9. N2 peak amplitudes (μV) in the left and right hemisphere for high and low anger participants. Data are expressed as mean (SEM)

4.3.2.3 NSW amplitude

To explore if RHCs modulated later cognitive control indices of attentional bias to threat in relation to trait anger, 2 (UHC group; RHC, LHC) x 2 (trait anger; high, low) x 2 (Word-type; threat, neutral) x 2 (Hemisphere; left, right) x 3 (Region; Frontal, frontocentral, central) mixed ANOVA was performed on NSW mean amplitudes. Descriptive statistics for NSW amplitudes are presented in Table 4.6 and inferential statistics for this analysis are presented in Table 4.7.

Table 4.6. NSW mean amplitudes (μV) at left and right frontal, frontocentral, and central sites for threat and neutral words in the RHC and LHC conditions for high and low anger participants. Standard deviations are in parenthesis

		RHC		LHC	
		High anger	**Low anger**	**High anger**	**Low anger**
		(N = 10)	***(N = 8)***	***(N = 10)***	***(N = 9)***
Threat Words	**F3**	-2.54 (2.39)	-1.93(2.58)	-2.81(3.57)	-2.89 (3.48)
	F4	-4.42 (3.74)	-1.48 (1.64)	-1.75 (2.38)	-2.92 (3.36)
	FC1	-7.67 (5.09)	-5.09 (6.87)	-2.32 (3.83)	-4.71 (3.74)
	FC2	-6.11 (4.31)	-3.90 (5.88)	-3.46 (2.86)	-4.55 (3.12)
	C3	-2.55 (2.16)	-2.01 (3.31)	-1.41 (1.91)	-1.66 (2.83)
	C4	-2.10 (3.53)	-1.17 (3.33)	-1.33 (2.10)	-1.15 (2.00)
Neutral Words	**F3**	-3.04 (2.64)	-2.35 (2.92)	-2.90 (3.36)	-2.41 (3.45)
	F4	-4.30 (3.94)	-1.43 (1.30)	-1.95 (2.81)	-2.96 (3.18)
	FC1	-7.46 (4.67)	-5.47 (6.95)	-3.37 (3.73)	-4.86 (3.87)
	FC2	-6.17 (3.94)	-3.96 (6.59)	-4.43 (3.52)	-4.75 (3.16)
	C3	-2.67 (2.23)	-1.86 (2.95)	-1.63 (2.21)	-2.22 (2.65)
	C4	-2.31 (4.00)	-1.36 (3.53)	-1.58 (2.31)	-1.40 (1.84)

Table 4.7. Inferential statistics for the effects of UHC, anger word, hemisphere and region on NSW mean amplitudes (μV). Significant effects are highlighted.

Condition	***F***	***p***
Anger	.29	.59
Hand	.68	.41
Word	4.20	.05
Word x UHC	.77	.39
Word x Anger	.54	.47
Word x UHC x Anger	.98	.33
Region	20.96	.000
Region x UHC	1.35	.27
Region x Anger	.05	.95
Region x UHC x Anger	.88	.42
Hemisphere	.90	.35
Hemisphere x UHC	.47	.50
Hemisphere x Anger	.67	.42
Hemisphere x UHC x Anger	.78	.60
Word x Region	1.56	.22
Word x Region x UHC	3.41	.04
Word x Region x Anger	.50	.61
Word x region x UHC x Anger	3.16	.052
Word x Hemisphere	.06	.80
Word x Hemisphere x UHC	.46	.50
Word x Hemisphere x Anger	.01	.98
Word x Hemisphere x UHC x Anger	.06	.81
Region x Hemisphere	.50	.57
Region x Hemisphere x UHC	2.80	.08
Region x Hemisphere x Anger	.01	.98
Region x Hemisphere x UHC x Anger	2.53	.10
Word x Region x Hemisphere	.24	.79
Word x Region x Hemisphere x UHC	3.82	.03
Word x Region x Hemisphere x Anger	.67	.52
Word x Region x Hemisphere x UHC x Anger	1.08	.35

In support of prediction 5, no main effect of UHC, anger, or hemisphere was found ($p > 0.05$, see Table.4.7). Contrary to prediction 5, the ANOVA revealed significantly smaller NSW amplitudes for threat than neutral words, F (1.00, 33.00) = 4.20, $p < .05$, np^2 = 11.This can be seen in Figure 4.10. A significant main effect of region was also found, F (1.79, 58.98) = 20.96, $p < .05$, np^2 = 39. Contrasts showed NSW was larger at frontocentral than frontal, F (1, 33) = 16.32, $p < .05$, np^2 = 33, and central sites, F (1, 33) = 37.17, $p < .05$, np^2 = 53. This can be seen in Figure 4.11.

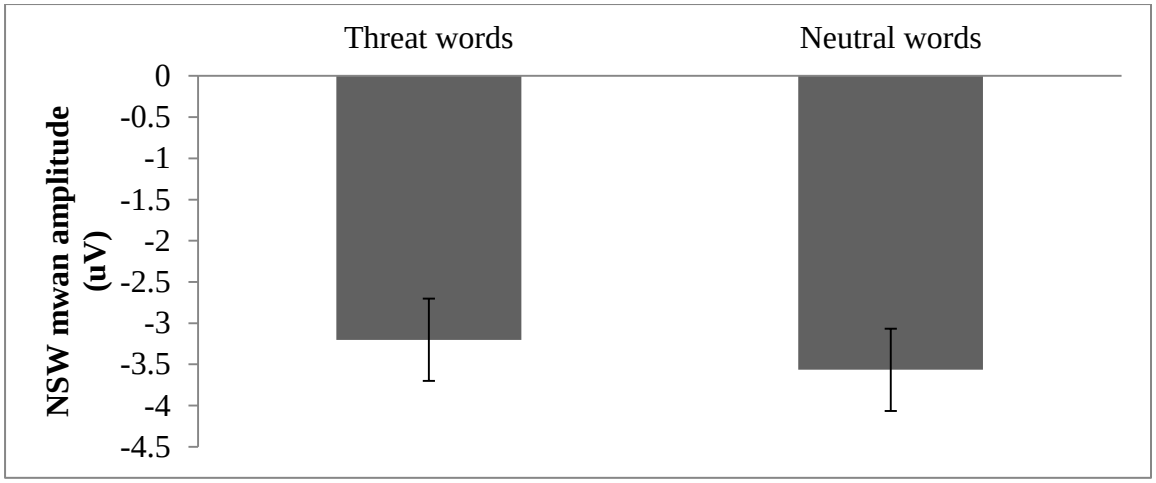


Figure 4.10. NSW mean amplitude (μV) for threat and neutral words in the EST. Data are expressed as mean (SEM)

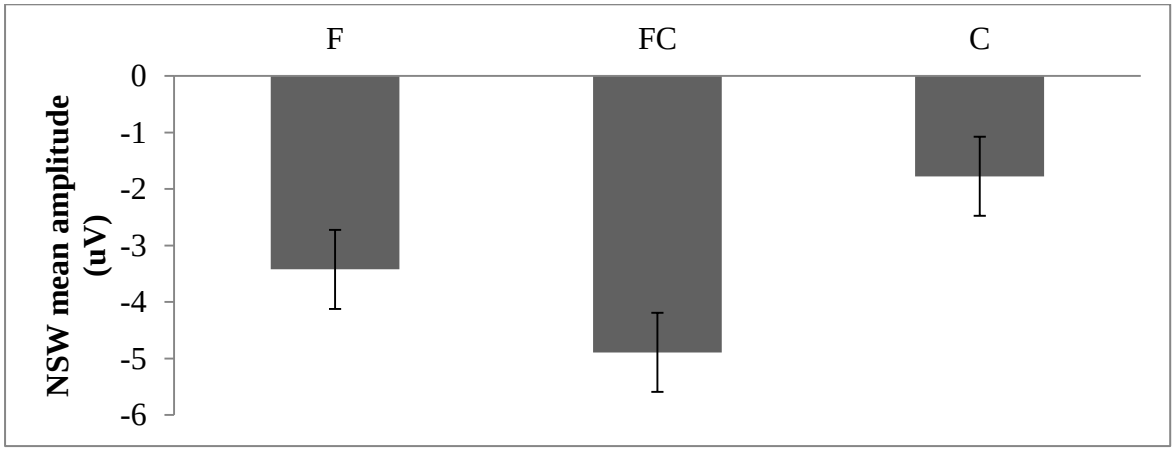


Figure 4.11. NSW mean amplitude (μV) at Frontal (F), frontocentral (FC) and central sites (C) during the EST. Data are expressed as mean (SEM)

With regards to prediction 6, the ANOVA showed no UHC × word interaction, (p > 0.05, see Table.4.7). However, the ANOVA did reveal a significant UHC × word × region interaction, F (1.91, 62.97) = 3.41, p <.05, np^2 = .09. Although, contrary to predictions, post hoc tests revealed that in the RHC group there was no NSW amplitude word-type difference at any regional sites (p > .05). In the LHC group there was also no NSW word-type amplitude difference at frontal or at central sites (p > .05) but at frontocentral sites NSW was significantly smaller for threat than neutral words, t (18) = 2.73, p = .01. This significant effect can be seen in Figure 4.12 and 4.13.

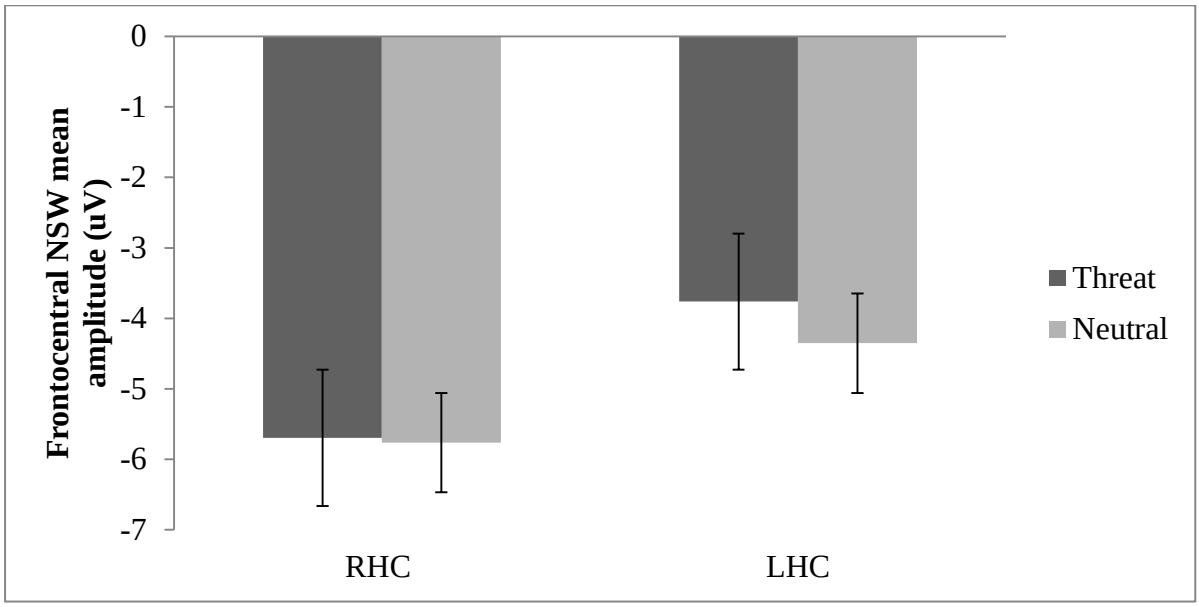


Figure 4.12. Frontocentral NSW mean amplitudes (μV) for threat and neutral words in the RHC and LHC groups. Data are expressed as mean (SEM)

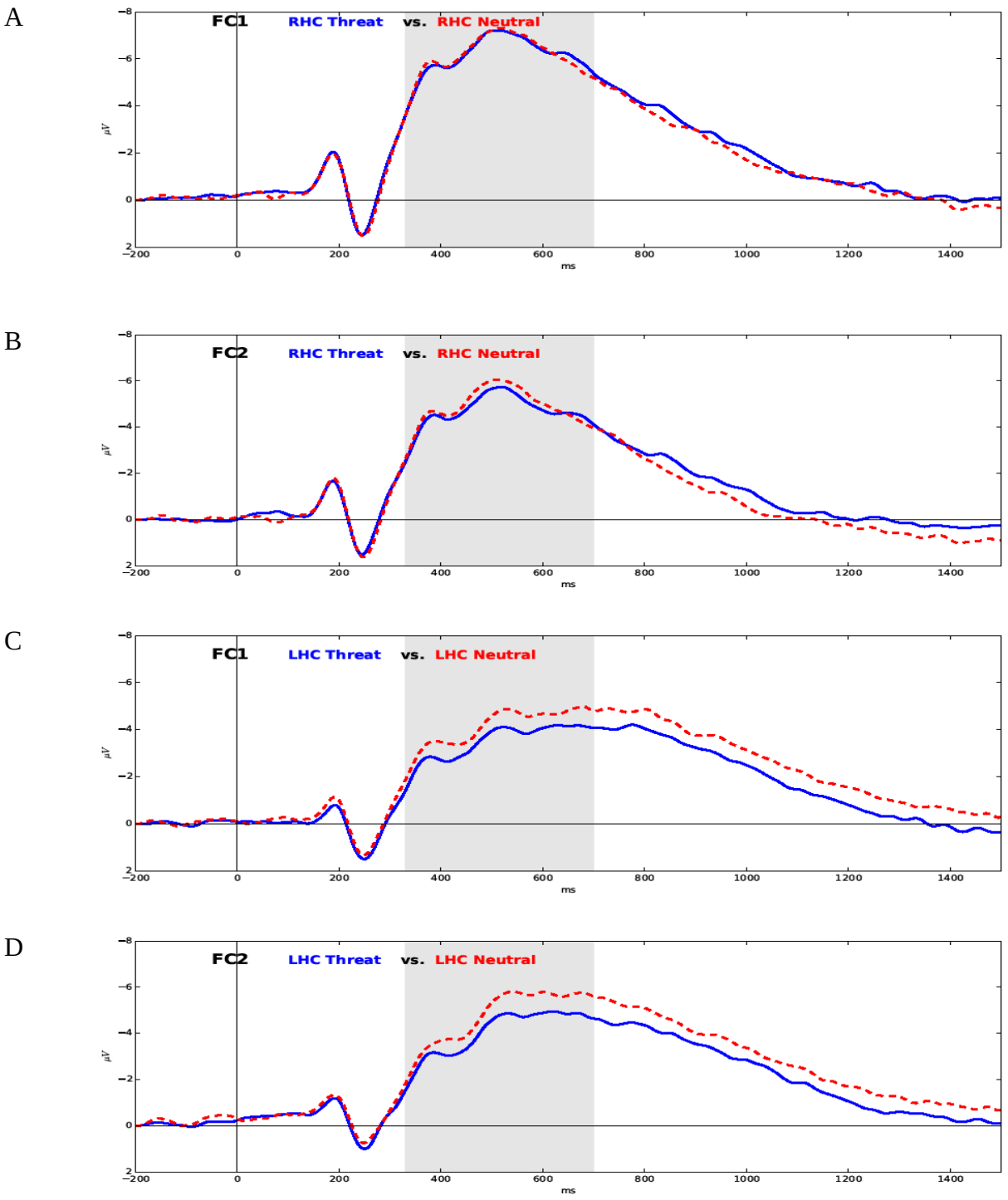
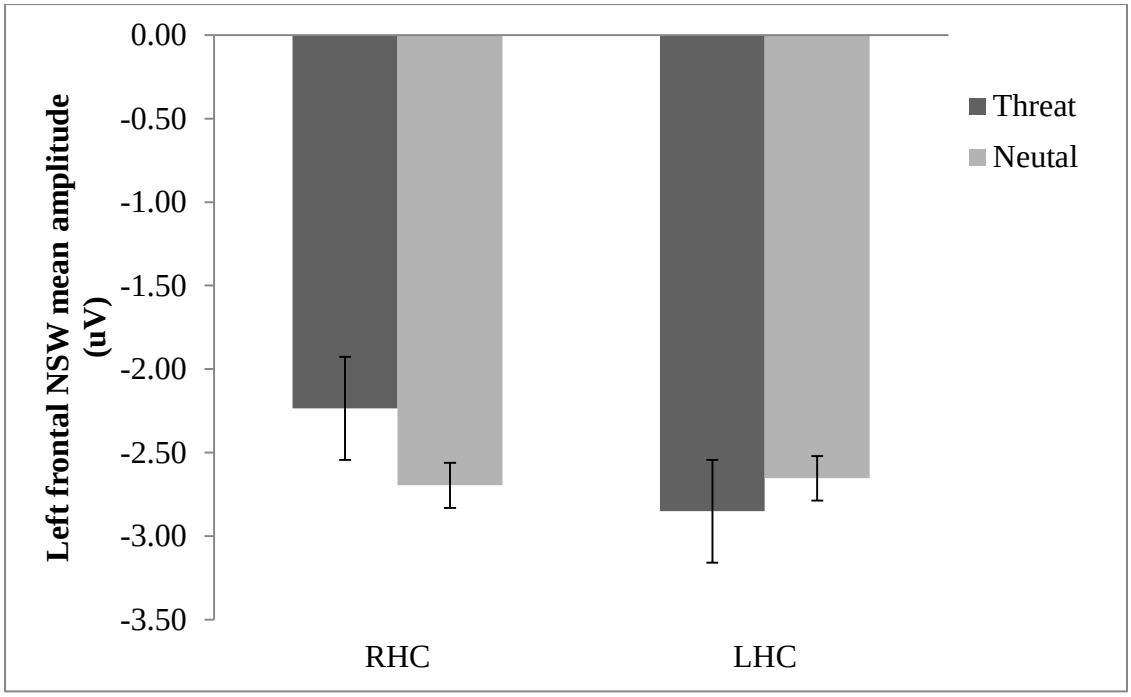


Figure 4.13. Grand average waveforms showing frontocentral NSW amplitudes (μV) (shaded grey) for threat and neutral words in the RHC (Panels A and B) and LHC group (Panels C and D). Shows significantly smaller frontocentral NSW for threat than neutral words following LHCs compared to RHCs

With further regards to prediction 6, the UHC × word × region interaction was qualified by a significant UHC × word × region × hemisphere interaction, F (1.99, 65.76) = 3.82, p <.05, np^2 = .10. Post hoc tests revealed that, in the RHC group there was no NSW amplitude word-type difference at frontocentral or central regions in the left and right hemispheres (p > .05). but at left frontal sites, NSW was significantly smaller for threat than neutral words, t (17) = 3.19, p < .01. This significant effect can be seen in Figures 4.14 and 4.15. In contrast, in the LHC condition, no NSW amplitude word-type difference was observed at left and right frontal or central sites, (p > .05) but NSW amplitudes were significantly smaller for threat than neutral words at both the left, t (18) = 2.20, p = .04**,** and right frontocentral sites, t (18) = 2.80, p = .01. However, following Bonferroni corrections, differences found in the LHC condition failed to reach significance.



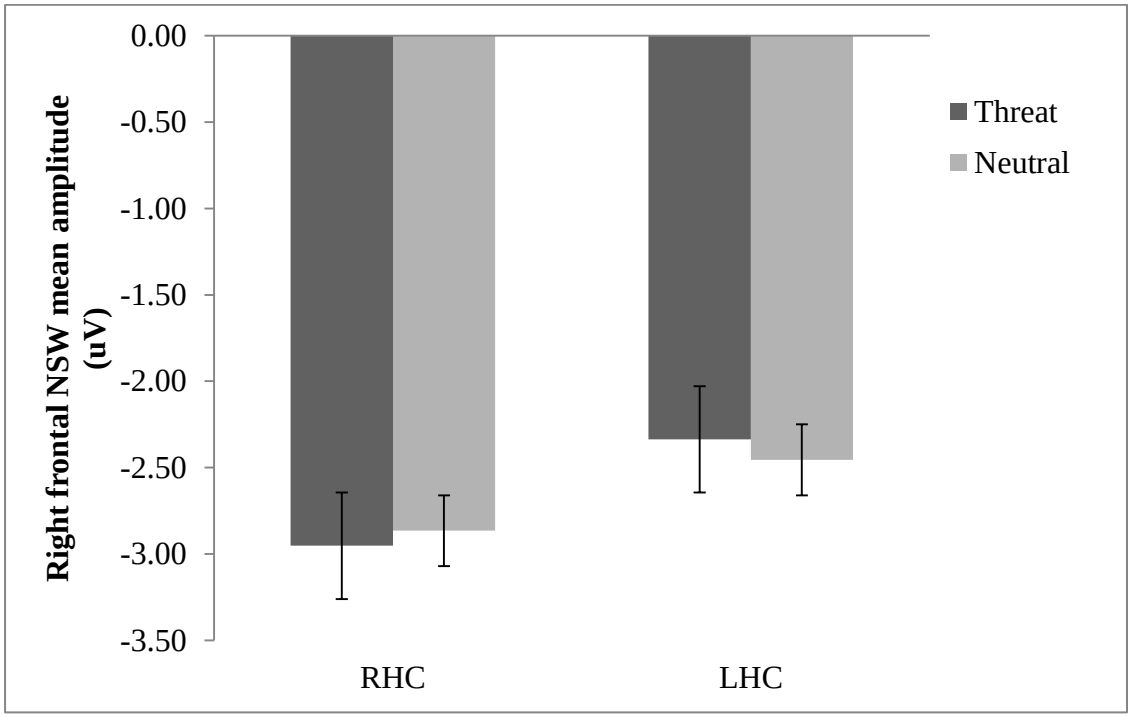


Figure 4.14. Mean frontal NSW amplitudes (μV) at left frontal (upper panel) and right frontal (lower panel) for threat compared to neutral words in the RHC compared to LHC groups

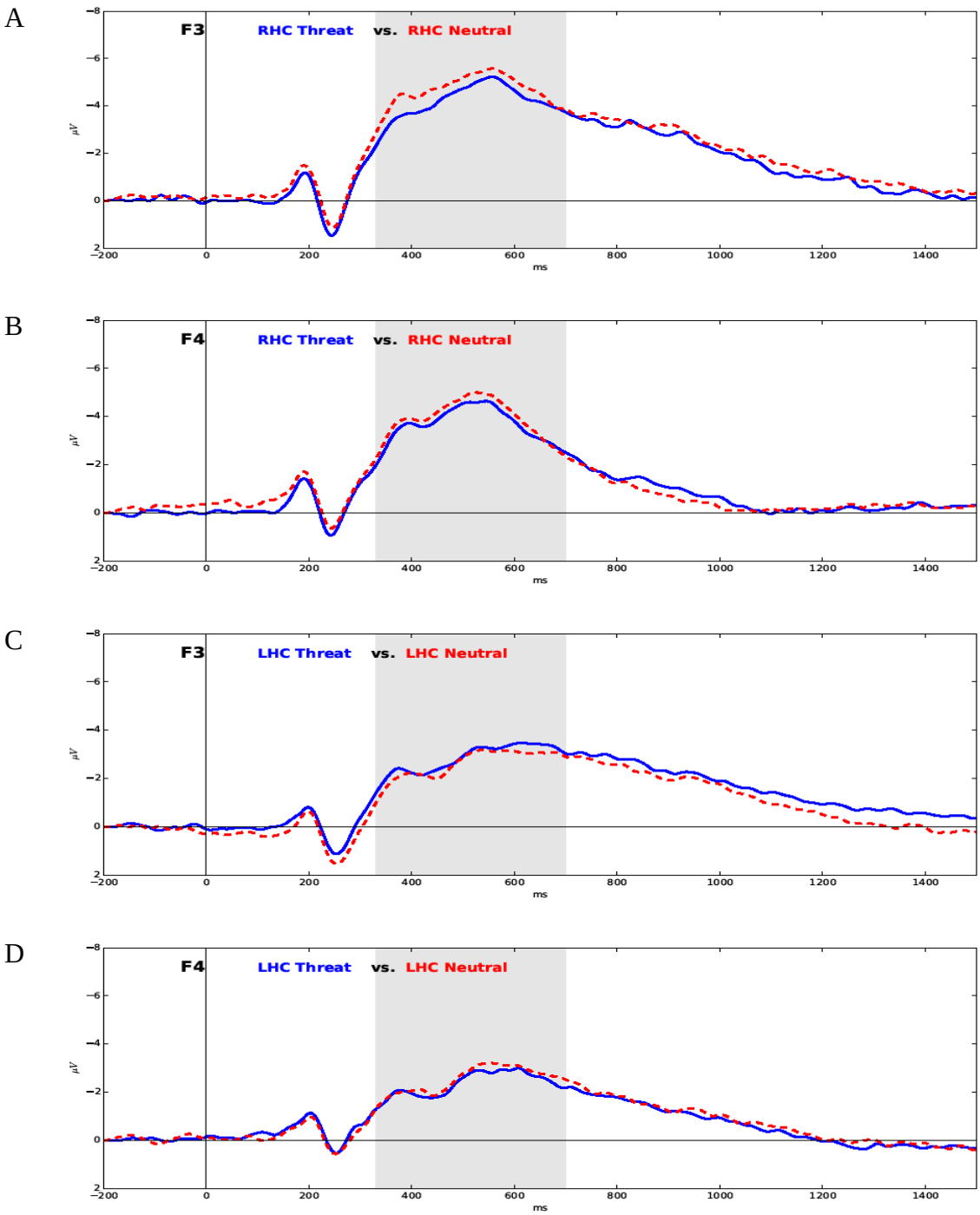


Figure 4.15. Grand average waveforms of frontal NSW amplitudes (μV) (shaded grey) for threat compared to neutral words at left and right frontal sites in the RHC (Panels A and C) compared to LHC groups (Panels B and D) respectively. Shows that at left frontal sites, RHCs compared to LHCs significantly reduced NSW for threat compared to neutral words

Contrary to prediction 7, the ANOVA showed no NSW amplitude anger × word interaction, found (p > 0.05, see Table.4.7). Furthermore, with respect to prediction 8, the ANOVA showed no UHC × anger × word interaction found (p > 0.05, see Table.4.7). However a trend emerged for a UHC × anger × word × region interaction, F (1.91, 62.97) = 3.16, p =.052, np^2 = .09. This showed that anger influenced the effect of the UHC in response to threat compared to neutral words. However, contrary to predictions post hoc tests revealed that in the RHC group, no NSW amplitude word-type difference was observed for both high and low anger participants, at any of the regional sites (p > .05). In the LHC group, no NSW amplitude word-type difference was observed at any regional site for low anger participants or in high anger participants at frontal or central sites (p > .05). However at frontocentral sites, NSW was significantly smaller for threat than neutral words, in high anger participants in the LHC group, t (9) = .48, p = .02, but following Bonferroni corrections these differences failed to reach significance. Nevertheless, this pattern of findings can be seen in figures 4.16, 4.17 and 4.18.

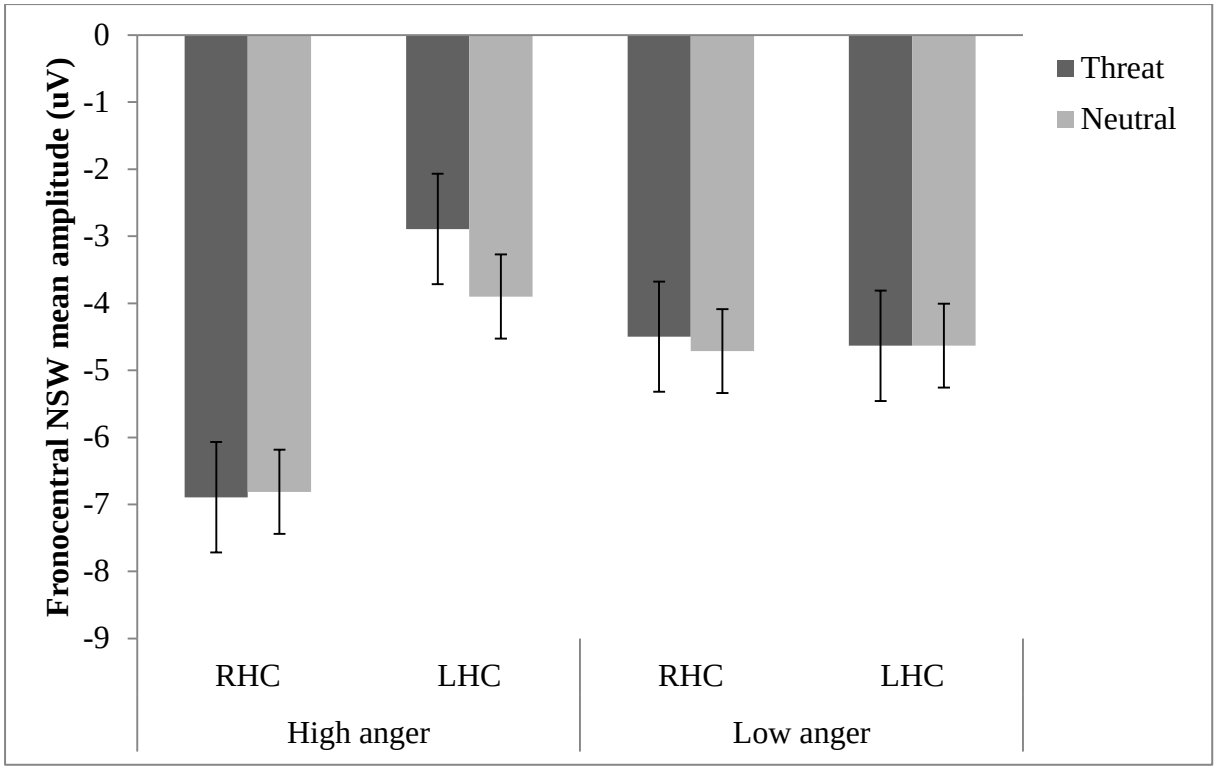


Figure 4.16. Mean frontocentral NSW amplitudes (μV) for threat compared to neutral words for high and low anger participants in the RHC compared to LHC group. Data are expressed as mean (SEM)

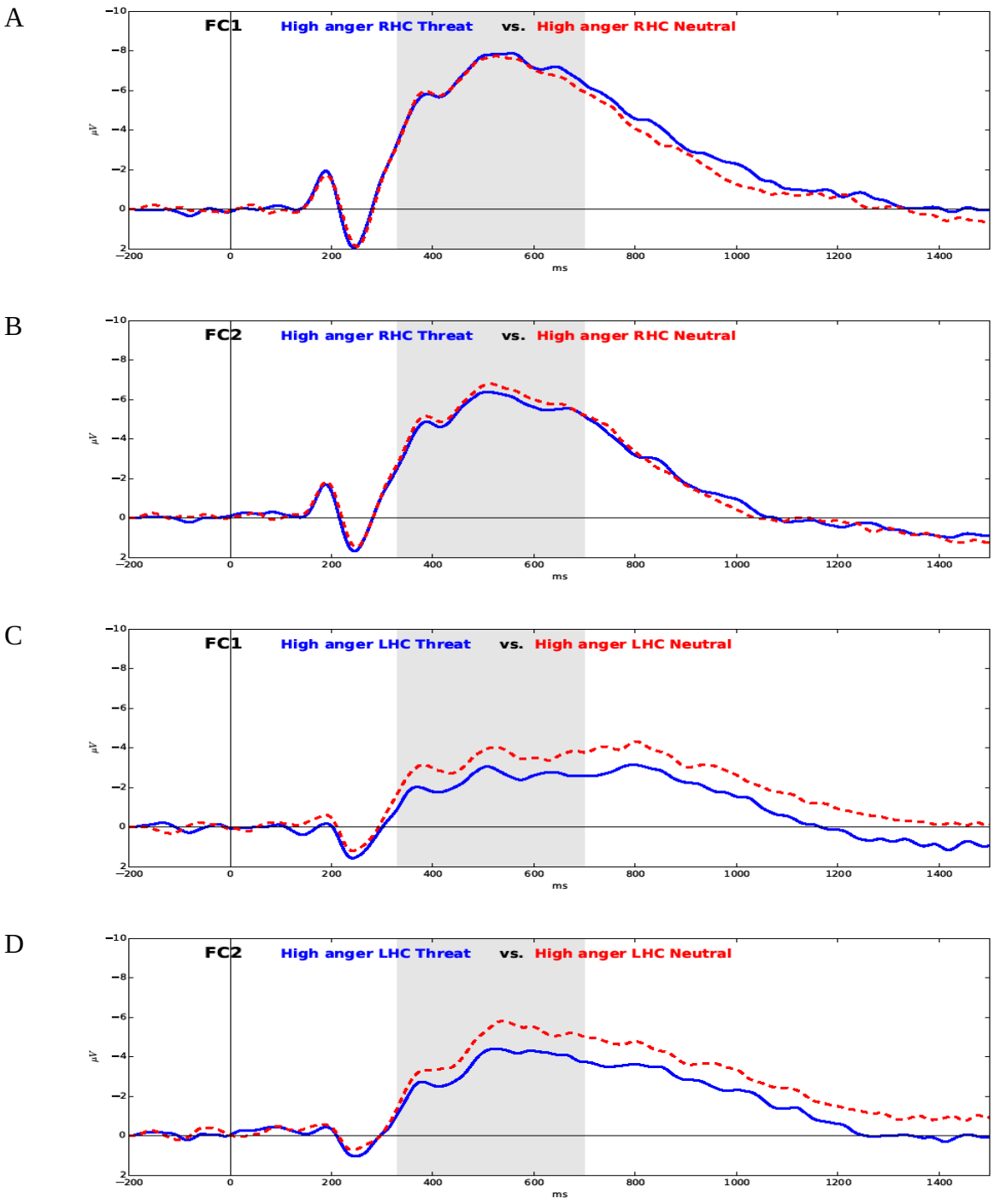


Figure 4.17. Grand average waveforms showing frontocentral NSW amplitudes (μV) (shaded grey) for threat compared to neutral words for high anger participants in the RHC (panels A and B) and LHC group (panels C and C). Shows a pattern where LHCs compared to RHCs reduced NSW for threat compared to neutral words

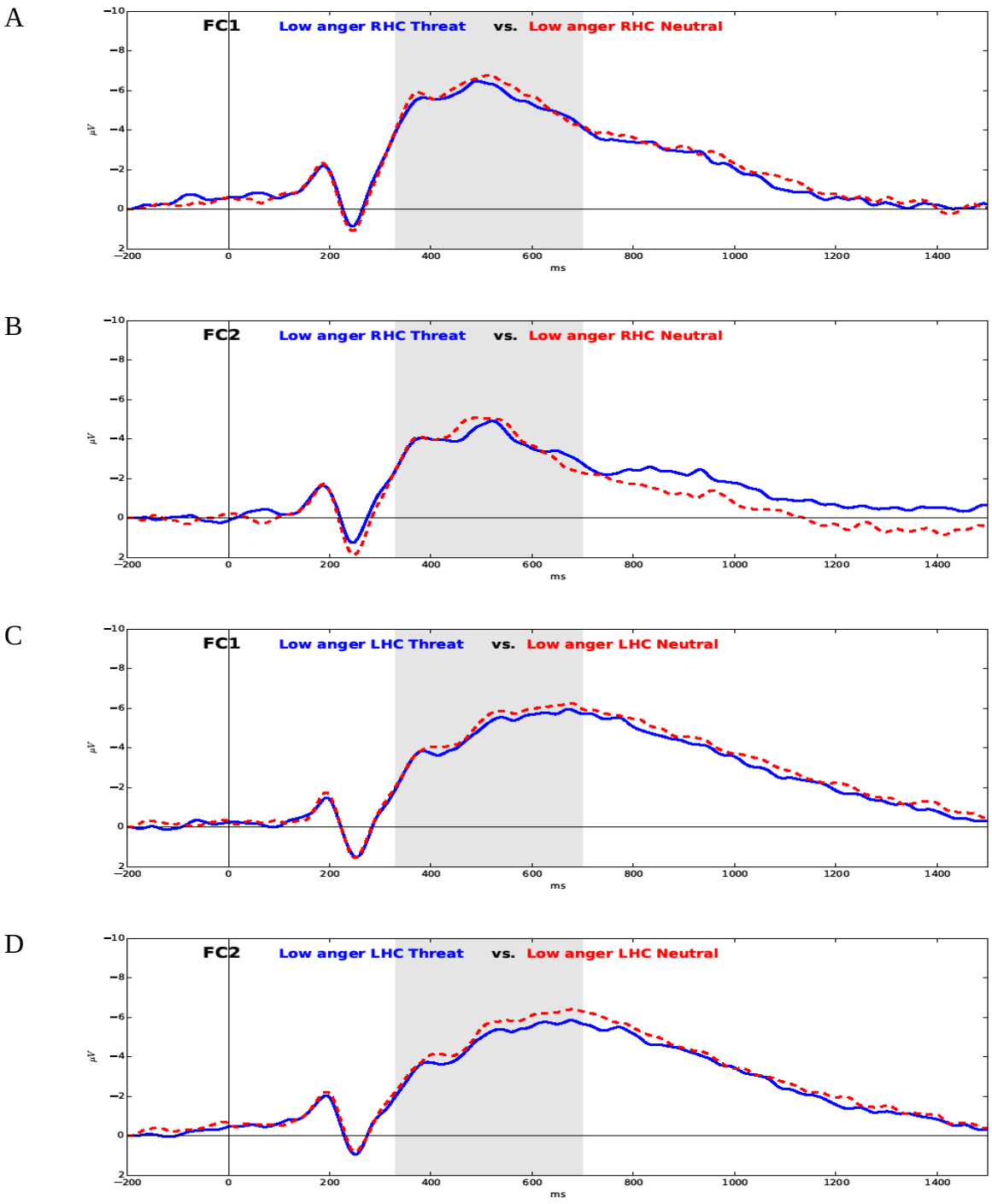


Figure 4.18. Grand average waveform showing frontocentral NSW amplitudes (μV) (shaded grey) for threat compared to neutral words for low anger participants in the RHC (panels A and B) and LHC group (panels C and C). Shows no effect of UHCs in response to word type

Contrary to predictions 9 10 and 11, the ANOVA showed no UHC × hemisphere interaction for NSW amplitude, anger × hemisphere interaction for NSW amplitude, and no UHC × anger × hemisphere interaction for NSW amplitude found ($p > 0.05$, see Table.4.7).. This indicated no NSW laterality effects of UHCs or anger in either isolation or in relation to each other was found.

4.4 Discussion

Study 2 integrated the EST paradigm with ERP indices of attentional bias, namely the N2, P3b and NSW to provide insight into whether the effects of the UHC modulations reflected motivational (P3b) or inhibitory (N2, NSW) aspects of attentional processing or both. Study 2 also explored the behavioural and neural effects of the UHC method in relation to trait anger. This Study provides evidence that that UHCs influence changes in attentional bias to threat. It also provides insight into the motivational and inhibitory mechanisms underlying these changes. The results also highlight how individual differences in trait anger influence the effect of the UHC method on attentional bias to threat.

4.4.1 Behavioural EST findings

As RHCs are known to induce approach motivation (Harmon-Jones 2006) and high anger (Peterson et al., 2008) and both have been associated with increased threat-related attentional bias in the EST (d'Alfonso et al. 2000; Smith and Waterman 2003), it was predicted that RHCs compared to LHCs and high compared to low anger would show increased attentional bias towards threat. Although whether it reflected motivational or control mechanisms remained unclear. These predictions were unsupported in the present study.

It was also hypothesised (prediction 4), in line with the Diathesis Stress Model (Davidson, 1992) that the effect of the RHC would be larger in high than low anger participants. The behavioural data from Study 2 (Chapter 4) showed that the effects of the UHC method were mediated by trait anger. However, contrary to predictions, RHCs did not increase attentional bias independent of trait anger or in relation to trait anger. Furthermore, LHCs did not modify attentional bias to threat in high anger participants. However, in low anger participants, LHCs increased threat-related interference. In line with the Diathesis Stress Model (Davidson 1992), it is possible that in low compared to high anger individuals (predisposed to greater relative right frontal brain activity) LHCs primed the activity of the right frontal brain, subsequently increasing negative affect and attentional bias to threat. It is also possible that LHC effects were not found in high anger as participants as these individuals are known to have greater relative left frontal brain activity (e.g. Harmon-Jones 2004) and therefore LHC priming effects on right frontal brain activity may not have been effective. This view is supported by evidence that has shown LHCs increase negative effect and bias perceptions and judgments negatively (Schiff and Lamon 1994). The finding are also supported by evidence showing an association between greater right frontal brain activity and attention bias towards threat (Perez-Edgar et al. 2013; Davidson 2004). As such the findings from the behavioural data are in line with a wealth of evidence that shows a positive association between negative affect and attentional bias towards threat (Bar-Haim et al. 2007).

The lack of threat-related attentional bias found for RHCs appears to be at odds with evidence that RHCs will increase approach motivation and anger (Harmon-Jones 2006; Peterson et al. 2008) and this should in turn increase threat-related interference (e.g. Smith and Waterman 2003). It is unclear at present why RHCs did not modulate behavioural measures of attentional bias to threat in relation to trait anger.

4.4.2 Summary of ERP findings

For brevity the following paragraphs will summarise the P3b, N2, and NSW findings in relation to predictions made separately. Following the findings in relation to the empirical and theoretical literature will be discussed.

4.4.2.1 P3b findings

P3b was explored to identify whether the facilitation effect found for RHCs in Study 1 reflected changes in motivational aspects of attentional processing of threat. In support of predictions that P3b amplitude would not be modulated by UHC, anger, word-type or hemisphere (prediction 5), no main effects were found in relation to the P3b component. However, contrary to predictions that relative to LHCs, RHCs would increase P3b amplitudes for threat compared to neutral words (prediction 6) no effect of word-type was found in the RHC group. Interestingly, when considering higher order interactions including region and hemisphere, while no P3b effects were found for the RHC group, the LHC group showed smaller P3b for threat than neutral words at left parietooccipital sites but this difference failed to reach significance following Bonferroni corrections. These findings suggest that the facilitation effect found in Study 1 did not reflect changes in motivational aspects of attentional bias to threat.

Contrary to predictions that P3b amplitude would be larger for threat compared to neutral words in high compared to low anger participants (prediction 7), no P3b effect was found. It was also predicted that relative to LHCs, RHCs would result in larger P3b for threat than neutral words and this effect would be larger in high than low anger participants (prediction 8). Contrary to predictions, higher order interactions including region showed that in the RHC group, high anger participants showed no P3b amplitude difference but low anger

participants showed larger P3b for threat than neutral words at centroparietal sites. In contrast, in the LHC group a reverse pattern was revealed with high anger showing larger P3b for threat than neutral words at centroparietal sites but low anger participants showing no P3b amplitude difference for word-type. However after Bonferroni corrections these findings failed to reach significance. Nevertheless this pattern of findings suggests that UHCs only modulate motivational aspects of attentional bias to threat in relation to anger. However given the lack of significance with this finding this interpretation is purely speculative.

It was also predicted that P3b would be larger in the left than the right hemisphere for RHCs compared to LHCs (prediction 9) and for high compared to low anger participants (prediction 10), and the effects would be largest for high anger participants in the RHC group (prediction 11). These predictions were not supported, no laterality differences were found for RHCs compared to LHCs, or for high compared to low trait anger, in isolation and in relation to one another.

4.4.2.2 N2 findings

N2 amplitudes were explored to identify whether the facilitation effect found for RHCs in Study 1 reflected changes in early cognitive control aspects of attentional processing of threat. Contrary to prediction 5, that there would be no main effects of UHC, anger, word-type, region or hemisphere a main effect of hemisphere was found. Results showed larger N2 in the left than the right hemisphere. This finding suggests that early cognitive control mechanisms in the left hemisphere were implemented during EST performance. This interpretation is consistent with the view that the left frontal brain plays a key role in the inhibition of emotion based responding (Davidson 2004).

It was also predicted that relative to LHCs, RHCs would increase N2 for threat compared to neutral words (prediction 6) and this effect would be larger in high than low anger participants (prediction 8). Contrary to predictions corresponding interactions were non-significant. However, higher order interactions including region revealed a trend in the LHC group independent of trait anger, where frontal N2 was larger for threat than neutral words but this difference failed to reach significance after bonferroni adjustments. It was also predicted that N2 would be larger for threat than neutral words in high compared to low anger participants (prediction 7). This prediction was not supported. It was also predicted that N2 would be larger in the left than the right hemisphere for RHCs compared to LHCs (prediction 9) and that this effect would be larger in high than low anger participants (prediction 11). These predictions were also not supported. These findings suggest that the facilitation effect found for RHCs in Study 1 did not reflect changes in early cognitive control mechanisms.

Furthermore contrary to predictions that N2 amplitudes would be larger in the left than the right hemisphere for high compared to low anger participants (prediction 10), high anger participants showed no N2 laterality difference whereas low anger participants showed larger N2 amplitudes in the right than the left hemisphere.

4.4.2.3 NSW findings

NSW amplitude was explored to identify whether the facilitation effect found for RHCs in Study 1 reflected changes in later cognitive control aspects of attentional processing of threat. Contrary to predictions that there would not be a main effect of word-type on NSW (prediction 5), results revealed significantly smaller NSW for threat compared to neutral words. It was also predicted that relative to LHCs, RHCs would increase NSW for threat compared to neutral words (prediction 6). This prediction was not supported. However,

higher order interaction including region showed that in the RHC group there was no effect of word-type on NSW amplitudes but in the LHC group, frontocentral NSW was significantly smaller for threat than neutral words. Importantly, when this higher order interaction effect was explored in relation to laterality, it emerged that the RHC group had significantly reduced NSW for threat compared to neutral words, at left frontal sites. This significant NSW amplitude effect suggests that the facilitation effect found for RHCs in Study 1, reflected changes in later cognitive control mechanisms. In the LHC group there was also a trend for smaller NSW for threat than neutral words at left and right frontocentral sites but following Bonferroni corrections this difference failed to reach significance.

The hypothesis that the effect of word-type on NSW would be larger in high compared to low anger participants (prediction 7) was not supported. Also contrary to predictions that relative to LHCs, RHCs would increase NSW for threat compared to neutral words and this effect would be larger in high than low anger participants (prediction 8) no significant effects were found. Similarly in the LHC group, low anger participants did not show a NSW difference for threat and neutral words. However, a pattern did emerge for high anger participants in the LHC group. They showed smaller frontocentral NSW for threat than neutral words. However, following Bonferroni corrections this finding failed to reach significance.

It was also predicted that NSW would be larger in the left than the right hemisphere for RHCs compared to LHCs (prediction 9) and for high compared to low anger participants (prediction 10), and the effects would be largest for high anger participants in the RHC group (prediction 11). These predictions were not supported.

4.4.2.4 Empirical and Theoretical interpretation of ERP findings

Collectively the ERP data from Study 2 further highlighted the effectiveness of the UHC to induce attentional changes in the threat processing and provide insight that the facilitation effect found for RHCs in Study 1 reflected changes in later cognitive processing of threat words but did not reflect changes in early cognitive control or motivational aspects of attentional bias to threat. Independent of UHC, there was no evidence of threat-related attentional biases in relation to anger. However, a threat-related attentional bias was produced by UHCs both independent of and in relation to trait anger.

Independent of trait anger, RHCs significantly reduced left frontal NSW for threat compared to neutral words. According to previous evidence (e.g. West and Alain 2000; van Hooff et al. 2008), this finding illustrated that RHCs had increased the efficiency of left frontal neural mechanisms implemented during the cognitive control of EST threat conflict. This supported predictions that RHCs would increase the inhibition of threat-related attentional bias and also provided insight into the facilitation effect found in Study 1 (Chapter 3). Furthermore, it provided direct support for the view that the left frontal brain plays a fundamental role in the inhibition of emotion based responding (Davidson 2004). Moreover, this went some way in explaining the lack of threat-related attentional bias found for RHCs in the behavioural data in Study 2. For instance, the increased neural efficiency of later control mechanisms may have prevented threat interfering with competing cognitive processes involved with task relevant responding. The lack of similar findings for N2 suggests that the effects of RHCs on changes to cognitive control are isolated to later inhibitory processing stages only.

Study 2 also showed that, RHCs effects were influenced by trait anger. A trend emerged in low anger but not high participants where RHCs increased centroparietal P3b for threat compared to neutral words. According to the ERP literature (e.g. Duncan-Johnson and Donchin 1977) these findings tentatively suggest that RHCs may have increased approach motivated task processing which prevented threat competing for attentional resources. However, this interpretation is purely speculative given the lack of significance of this finding. Nevertheless, this interpretation is consistent with evidence that has shown RHC's prime the approach motivational system (Harmon-Jones 2006) and provided further support for the motivational hypothesis (e.g., Davidson and Irwin 1999; Davidson et al. 2000). However, this finding is somewhat inconsistent with evidence that has shown increased left frontal brain activity elicits approach motivated attentional bias towards threat, resulting in threat-related interference (d'Alfonso et al. 2000). However, methodological differences between rTMS and UHC methods used to modulate frontal brain activity, (discussed in depth in Chapter 3) and the use of ERP data may explain the discrepancy between findings. Furthermore, it is important to note that a lack of significance in the RHC findings make any inferences drawn purely speculative at present. Nevertheless, the finding from Study 2, suggest that RHCs modulated motivational aspects of attentional processing in relation to trait anger and that independent of anger, RHCs modulated later cognitive components of attentional processing.

Unexpectedly, some interesting trends also emerged for LHCs both in isolation of and in relation to trait anger. In isolation of trait anger, LHCs enhanced N2 amplitudes and reduced both left parietooccipital P3b and right frontocentral NSW for threat compared to neutral words. According to N2 evidence (e.g. Dennis and Chen 2007, 2009; Van Veen and Carter 2002) these findings indicate that LHCs reduced the efficiency of early ACC cognitive

control mechanisms required to inhibit EST interference. The reduced parietooccipital P3b indicates that following LHCs, threat also becomes more motivationally relevant than neutral words and competed more for attentional resources (Duncan-Johnson and Donchin 1977; MacNamara, et al. 2009). Furthermore, the reduced NSW indicated that less cognitive control was recruited to resolve threat conflict at later inhibitory stages (e.g. West and Alain, 2000; van Hooff, et al. 2008). However, the lack of significance of these LHC findings means that the following interpretations are purely speculative without further investigation. Nevertheless, this pattern of findings may suggest that LHCs increased threat-related attentional bias at all cognitive processing stages. These interpretations of the LHC effect are harmonious with previous UHC literature, which has shown LHCs induce negative affect (Schiff and Lamon 1994). Therefore, it was possible that in isolation of anger, LHCs increased negative affect and motivational relevance of threat which subsequently increased the allocation of attentional resource towards threat and reduced the capacity to recruit attentional control over threat.

These assumptions are consistent with the Valence hypothesis (e.g. Davidson 1984) that posits that the right frontal brain is associated with negative emotions and with evidence of a positive association between negative affect and threat-related attentional bias (Bar-Haim et al. 2007). Alternatively, these findings are also consistent with the motivational hypothesis that postulates that right frontal brain activity is positively associated with withdrawal motivation (e.g. Davidson and Irwin 1999; Davidson et al. 2000) and with evidence that withdrawal motivation increases attentional bias towards threat (Gray 1994). Therefore, it is possible that LHCs increased activity of right withdrawal networks resulting in inhibited task responding in favour of greater allocation of attention towards threat. However, it is important to note that the present LHC findings make it impossible to disentangle the effect

of valence and motivation on hemispheric specialisation. This further illustrates the usefulness of exploring anger (a negative approach motivated emotion) for investigating whether frontal brain asymmetry patterns reflect motivation or valence in attentional bias research.

In Study 2, findings also showed that the effects of LHCs interacted with trait anger. A trend emerged in high anger participants where LHCs increased centroparietal P3b and reduced frontocentral NSW for threat compared to neutral words. According to previous ERP literature, these findings suggested that in high anger participants, LHC's increased the motivational processing of threat increasing task difficulty (e.g. Johnston et al. 1986; MacNamara et al. 2009) and reduced the cognitive control recruited to resolve the subsequent threat conflict (e.g. West and Alain 2000; van Hooff et al. 2008). These findings indicated that in high anger participants that are already susceptible to preferential threat processing, LHCs further increased attentional bias to threat. This assumption is supported by the lack of attentional bias to threat found in Study 2 for trait anger, in isolation of UHCs. Therefore, this further highlights the usefulness of the UHC method when exploring attentional bias to threat in healthy samples.

4.4.2.5 Further considerations

While the findings from Studies 1 and 2 provided novel evidence that the UHCs modulates the extent that threat interferes with competing cognitive processes, they did not provide insight into which aspect of attention allocation (e.g. vigilance, avoidance or disengagement processes) was influenced by the UHC (MacLeod, et al., 1986).

As discussed in Chapter 1 and 2, the EST paradigm involves the use of competing stimuli presented in the same spatial location and as such cannot provide insight into whether the increased neural efficiency found in the RHC group would modulate control over early vigilance or later disengagement from threat. Establishing which attentional component the UHC method modulates would provide more fine grained insight into the causal relationship between approach motivation and attentional bias to threat. As discussed in Chapter 2, the dot-probe task paradigm (DPT; MacLeod, et al, 1998) allows individual componential characteristics of attentional bias to be explored. The issues discussed were addressed in Studies 3 and 4 (Chapters 5 and 6).

Furthermore, the current study did not include positive words to explore the effect of RHC on attentional bias to threat. For this reason, emotional valence may be confounded with emotional arousal. Given that models of emotion classify emotion along two basic dimensions, valence and arousal (e.g., Barrett 2006; Lang, Bradley, and Cuthbert, 1990) it is important to explore the contribution of both dimensions on attentional bias. Research that has matched levels of arousal have shown attentional bias towards emotionally arousing stimuli rather than to threat-related stimuli per se (e.g., Fischler and Bradley, 2006; Herbert, Junghöfer, and Kissler, 2008; Kanske and Kotz, 2007). These issues were also addressed in Studies 3 and 4 (Chapters 5 and 6).

The findings of Study 2 warranted further exploration of how the UHC method modulates attentional bias to threat in subclinical populations. As questions remain about the componential and word-type characteristics of the UHC effects, these were explored in the

next Chapter using DPT as a measures of attentional bias which included threat, positive and neutral word-types while controlling for trait anger.

CHAPTER 5

STUDY 3: ATTENTIONAL PROCESSING OF EMOTIONAL WORDS: EFFECTS OF LATERALIZED HAND CONTRACTIONS

5.1. Introduction

The aim of the research presented in this Chapter was to explore which components of attentional bias were modulated by the facilitation effect found for RHCs, in Study 1 (Chapter 3) and the increased neural efficiency of cognitive control mechanisms found for RHCs, in Study 2 (Chapter 4). This is important in order to understand whether the increased efficiency of the inhibitory mechanism induced via RHCs, would be reflected in changes in early vigilance or later disengagement difficulties. Given the lack of research in this field, an investigation of the componential characteristics of the UHCs effect on attentional bias to emotion related words was warranted to build an understanding of this method when used with a DPT paradigm.

As discussed in Chapter 1, attentional bias is an underlying mechanism of the fear system that serves a normative function that prioritises threat over positive and neutral information there allowing rapid detection of and appropriate responding to threat in the environment (e.g. Öhman 2005; Bar-Haim et al. 2007). However, this normative attentional bias to threat is further enhanced when threat elicits emotional states (Öhman 2005) such as anger. For this reason, individuals who have high trait anger and are therefore more prone to state anger are likely to have an enhanced attentional bias to threat (e.g. van Honk et al. 2001). This non-normative attentional bias is also implicated in the occurrence of aggressive behaviour

(Todorov and Bargh 2002; Smith and Waterman 2003). Therefore, it is important that a greater understanding is gained with regard to attentional bias to threat in relation to anger.

It is also important to note that evidence has emerged showing that attentional bias is related to emotional arousal rather than valence (e.g. Kanske and Kotz 2007; Fischler and Bradley 2006; Herbert et al. 2008). As such, it is uncertain whether anger-related attentional bias reflects preferential processing of general highly arousing information or threat per se. As discussed in Chapter 4 (Study 2), because positive words were not included to explore the effect of RHCs on attentional bias to threat, valence and arousal may be confounded. As such, it was difficult to establish whether the attentional changes observed in Studies 1 and 2 reflected greater efficiency of control over threat or arousal. For this reason, Study 3 also aimed to explore if the RHC effects found in Studies 1 and 2 were related to valence or high arousal words in general. In order to establish whether attentional bias modifications were a result of attentional changes to valence or arousal Study 3 examined the effect of UHCs on attentional bias to high arousing emotional relative to low arousal neutral words. This would provide an index of arousal. The study also explored attentional bias towards high arousal threat words presented with equally high arousing positive words. This would provide an index of true attentional bias to threat. This exploration will provide insight into whether UHCs modulate attentional bias in relation to general arousal or to threat specifically.

While a handful of studies have shown that attentional bias to threat is indeed related to high anger (e.g. van Honk, Tuiten, de Hann et al. 2001), these studies leave unanswered questions. Firstly, as these studies used only threat stimuli, they provide no insight into whether anger-related attentional bias reflected a bias towards high arousal or threat valence. Secondly,

these studies employed the EST paradigm (Williams et al. 1996), which involves the use of competing stimuli presented in the same spatial location. As discussed in Chapter 2, the EST has received criticism (MacLeod et al. 1986) as it cannot differentiate the componential characteristics of attentional bias i.e. facilitated attention towards threat, difficulty disengaging from threat (attentional capture) and attention avoidance of threat. As such, these EST studies provide no insight into the componential characteristics of attentional bias to threat. Similarly, as the EST was employed in Study 1 and 2, previous findings reported in this thesis cannot provide insight into whether the increased facilitation found in Study 1, or the increased neural efficiency found for RHCs in Study 2, was a result of increased cognitive control over early vigilance or later disengagement components of attentional bias to threat.

In contrast, as discussed in Chapter 2, the DPT (MacLeod, Mathews, and Tata 1986) allows a detailed exploration of these attention components. In the DPT, participants view emotion-neutral word-pairs and RTs are recorded to probes at emotion congruent or incongruent locations. Faster congruent than incongruent RTs reflects vigilance whereas slower congruent than incongruent RTs reflects avoidance. However, following concerns about whether congruency effects reflected vigilance or disengagement difficulties the DPT was modified to include neutral-neutral baselines (Koster et al. 2004). This demonstrated faster emotion congruent than baseline RTs reflect vigilance whereas slower emotion incongruent than baseline RTs reflect disengagement difficulties. The incorporation of baselines have now indicated that attentional bias to threat frequently reflects later disengagement rather than early vigilance processes in relation to negative affect (Cisler et al. 2009).

To date, only one study has employed the DPT to explore anger-related attentional bias to threat (Smith and Waterman 2003). This study revealed a relationship between aggression and attentional bias towards threat. However, it is important to note that Smith and Waterman did not report analysis to identify whether attentional bias towards threat reflected vigilance or later disengagement difficulties. Therefore, it remains unclear which mechanisms of attentional bias are influenced by anger. Furthermore, the Smith and Waterman study used only threat words in a correlational design. This has led to uncertainty about the causal relationship between components of attentional bias to threat and anger.

As discussed extensively in previous chapters, exploring how the UHC method modulates attentional bias to threat may provide a better understanding of this causal relationship. The UHC literature has shown that RHCs increase left frontal brain activity which primes approach based motivational systems (Harmon-Jones 2006) and induces aggression (Peterson et al. 2008). This evidence supports motivational models of frontal brain asymmetry (e.g. Davidson 1995) in that increased approach motivation will guide and motivate goal relevant behaviour (for a review see, Harmon-Jones et al. 2010). Previous UHC evidence and motivational models would suggest that RHCs should increase approach motivation and subsequently increase the attentional resources allocated towards threat-related stimuli.

This assumption is supported by research that has shown a positive association between left frontal activity and approach-motivated attentional bias towards threat (interference) (e.g. d'Alfonso et al. 2006). Findings from these studies suggest that, approach motivated attentional bias may reflect a reduced ability to inhibit threat processing during competing tasks. However, this assumption is inconsistent with the pattern of findings in Studies 1 and

2, where increased facilitation and neural efficiency of later cognitive control mechanisms were found following RHC's (see Chapters 3 and 4). This assumption also challenges the view that the left frontal brain plays a fundamental role in the inhibition of emotion based responding (Davidson 2004). According to this view, increased left frontal brain activity should reduce attentional bias towards threat and increase task relevant processing. Indeed, results from the studies presented in this Thesis so far support this view.

The present study aimed to explore the effects of UHCs on attention bias to emotion words in a modified DPT (Koster et al. 2004). As increased attentional bias to threat is related to trait anger (e.g. van Honk et al 2001) this was controlled for using the AX-O subscale of the STAXI-2 (Spielberger 1999). Given the novelty of this research, controlling for trait anger ensured that any observed effects in the DPT paradigm could be confidently attributed to the UHCs and not trait anger.

5.1.1. Predictions

Four predictions were made in the present study. Which are as follows:

1. Based on the lack of main UHC effects in Studies 1 and 2, it is predicted that in isolation of word-type, there will be no main effect of UHC on DPT responses.
2. Based on evidence that all humans show attentional bias to threat over positive and neutral information (e.g. Öhman 2005), it is predicted that there will be an attentional bias for threat compared to positive and neutral words, independent of UHCs. This will be indexed by faster responses to threat than positive or neutral congruent compared to incongruent probes.

3. Based on findings from Study 1 and 2 and in line with evidence, that greater left frontal brain activity increases cognitive control over emotional responding(Davidson, 2003), it is predicted that compared to LHCs and NHCs, RHCs will reduce attentional bias to threat compared to positive and neutral words. This will be indexed by equal responses to threat compared to positive and neutral congruent than incongruent target in the RHC compared to LHC or NHC condition.
4. Based on evidence that attentional bias to threat reflects disengagement processes rather than vigilance (Cisler, et al., 2009). It is predicted that reduced attentional bias to threat versus positive and neutral words for RHCs relative to LHCs and NHCs will reflect reduced disengagement difficulty rather than reduced vigilance. This will be indexed by slower threat incongruent than baseline RTs.

5.2. Method

5.2.1. Design

To examine the effects of UHCs on attentional bias to emotion-related words the study employed a 3 (UHC condition, RHC, LHC, NHC) x 4 (word-pair, threat-neutral (T-N), threat-positive (T-P), positive-neutral (P-N), neutral-neutral (N-N) x 2 (probe position, Congruent, Incongruent) repeated measures design. There were 2 DVs. To explore if UHCs increased attentional bias to threat compared to positive and neutral words the dependent variable was DPT bias scores. To explore whether attentional bias to threat versus positive words reflected vigilance or disengagement difficulties the dependent variable was DPT RTs (ms). Information about the transformation of RTs to bias score and information on calculating whether attentional bias reflects vigilance of disengagement difficulties can be found in Sections 2.5.2.1 and 2.5.2.2 respectively.

5.2.2. Participants

An opportunity sample of 29 undergraduate Coventry University students was recruited for Study 3 from the sampling phase of the study. For information on the sampling phase of the study see Section 2.2. After data screening, three participants' data were excluded due to excessive missing and erroneous responses. Data from 26 participants (23 females; 3 males) aged 18 to 47 (*M* =19.7, *SD* = 5.62) were included in the final analysis. Participants had a trait anger score of, *M* = 17.96, *SD* = 5.35, indexed by the State-Trait Anger Expression Inventory STAXI-2 (Spielberger 1999) which fell within the 50th percentile of the trait anger scale. All participants also met the inclusion criteria and ethical guidelines were adhered to (described in section 2.2.).

5.2.3. Materials

5.2.3.1. Word stimuli

Information about the word stimuli for this study can be found in Section, 2.5.4. The list of words used in Study 3 is also presented in Appendix 2.

5.2.3.2.The State-Trait Anger Expression Inventory STAXI-2

Trait anger was assessed using the AX-O subscale of the revised 57-item State-Trait Anger Expression Inventory STAXI-2 (Spielberger 1999). For information about this metric, see section 2.3.2.

5.2.3.3. The Dot probe task (DPT)

Information about the DPT experimental protocol used in this and the next study (Chapter 6) are presented in detail in Section 2.5.3.

5.2.4. The UHC method

Information about the UHC procedure is presented in detail in section 2.4.

5.2.5. Procedure

Before the DPT, participants provided informed consent, and then completed the DPT in all UHC conditions in a single session. Participants completed UHCs immediately prior to task onset. Latin squared counterbalancing dictated which UHC type (RHC, LHC, NHC) was performed before each DPT block onset. During the DPT participants responded as quickly as possible to the probe location through a button press. Each session lasted 70 minutes, with 5-minute breaks between blocks.

5.3. Results

5.3.1. Data preparation

Errors and trials with RTs < 200ms or > 1500ms and greater than two SD from the mean were omitted. The mean percentage of data lost was 2.6 %. Bonferonni corrections were applied and an alpha level of 0.05 was used for all statistical tests unless stated otherwise. Significant interactions were explored using repeated measure contrasts.

5.3.2. DPT bias score analysis

To assess the effect of UHCs on attentional bias to emotion words, bias-scores were entered into a 3 (UHC condition; RHC, LHC, NHC) x 3(word-pair; TN, PN, TP) repeated measures analysis of covariance (ANCOVA). As attention bias is strongly correlated to TA (e.g. van Honk et al., 2001), AX-O scores (M= 17.96; SD = 5.3) were treated as covariate thereby

allowing a more direct examination of the UHC effects. Descriptive statistics for each condition are shown in Table 5.1. Data were within acceptable levels for normal distribution. Mauchly's test showed sphericity was assumed for main effect of word-pair, χ^2 (2) = .46, p > .05, UHC condition χ^2 (2) = 5.7, p> 0.05 and hand x word pair interaction χ^2 (9) = 9.63, p > 0.05.

Table 5.1. Mean DPT bias scores for TN, PN and TP word-pairs in the RHC, LHC and NHC conditions (standard deviations are shown in parentheses). Positive scores reflect vigilance and disengagement difficulties and negative scores reflect avoidance.

UHC	**TN Bias scores**	**PN Bias scores**	**TP Bias scores**
	M	*M*	*M*
RHC	1.56 (10.35)	-0.78(14.27)	-3.21 (13.14)
LHC	7.02 (16.73)	-1.17 (16.66)	2.00 (19.82)
NHC	2.07 (17.38)	4.03 (17.94)	1.05 (13.15)

As hypothesised in prediction 1, analysis revealed no DPT bias-score difference for trait anger, F (1, 24) = 1.20, p = .28, ηp^2 = .048, r = .22, or UHC condition, F (2, 48) = .210, p = .811, ηp^2 = .009, r = .06.

In line with prediction 2, analysis revealed a significant effect of word-pair, F (2, 48) = 4.48, p < .05, ηp^2 = .157. Planned contrasts showed more positive bias-scores for TN (M = 3.55, *SE*

= 1.60) than TP bias-scores (*M* = -.055, *SE* = 1.60), *F* (1, 24) = 9.52, *p* = .005, η^2 = .28, , indicating that regardless of UHC condition, attentional bias was greater for threat combined with neutral words than for threat combined with positive words. Contrasts also revealed significantly more negative TP than PN (*M* = .69, *SE* = 1.78) bias-scores, *F* (1, 24) = 4.50, *p* = .044, η^2 = .16, indicating no clear attentional bias for TP but an attentional bias towards positive words when these were combined with neutral ones. Contrast also revealed that TN and PN bias-scores were not significantly different, *F* (1, 24) = .52, *p* = .48, indicating that both produced attentional bias towards emotional words regardless of valence.

In support of prediction 3, analysis revealed a significant word-pair × hand interaction, *F* (4, 96) = 2.65, *p* < 0.05, ηp^2 = .10, independent of trait anger, *(4, 96) = 2.11, p = .09,* ηp^2 *= .09*. Contrast revealed more positive TN bias-scores in the LHC than the NHC condition and more negative PN bias-scores in the LHC than the NHC condition, *F* (1, 24) = 6.33, *p* < .05, ηp^2= .21. Contrast also showed that TN bias-scores were less positive in the RHC than the LHC condition but PN bias-scores were similar in the RHC and LHC conditions, *F* (1, 24) = 6.80, *p* < .05, η^2 = .22. RHC and NHC conditions did not differ when comparing TN with PN, *F* (1, 24) = .65, *p*=.43, PN with TP, *F* (1, 24) = .16, *p* >.05, or TN with TP bias-scores, *F* (1, 24) = .19, *p* > .05. No differences were observed between the RHC and LHC conditions for PN compared to TP bias-scores, *F* (1, 24) = 2.37, *p* >.05 or between TN and TP, *F* (1, 24) = 1.36, *p* >.05. No differences were found between LHC and NHC conditions for TN compared to TP bias-scores, *F* (1, 24) = 1.25, *p* >*.05*, or for PN compared to TP trials *F* (1, 24) = 2.62, *p* >*.05*. This pattern can be seen in Figure 5.1.

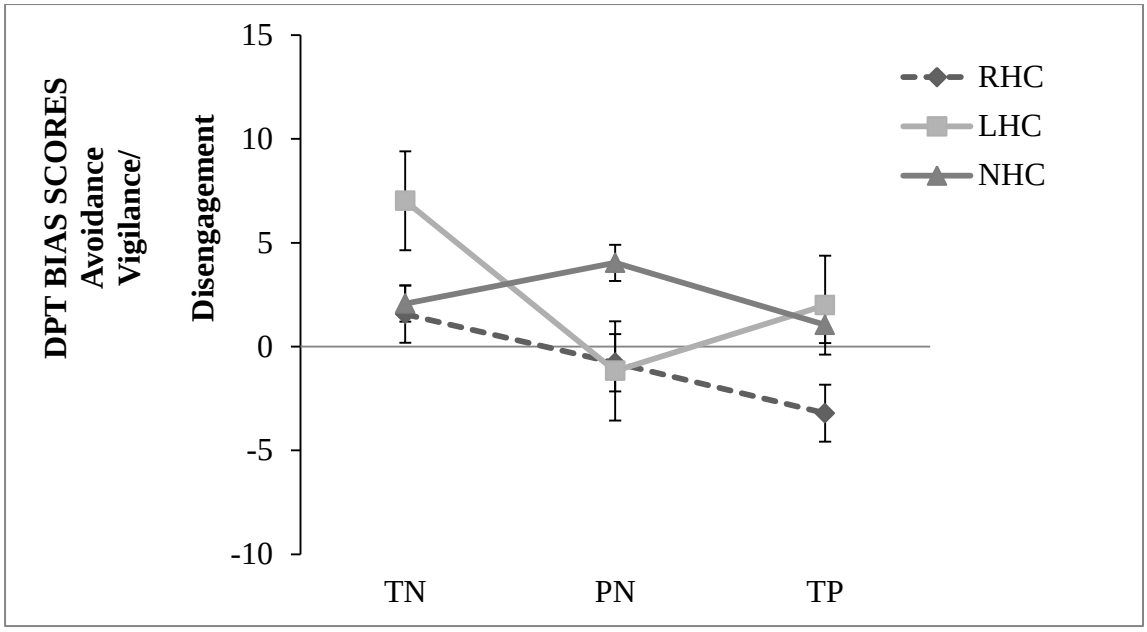


Figure 5.1. Mean DPT bias-scores for the TN, PN and TP word-pairs in the RHC, LHC and NHC condition. NN trials are not included in the initial analysis, as congruence type cannot be differentiated. Positive bias-scores reflect faster congruent than incongruent responses (vigilance or disengagement difficulties). Negative bias-scores reflect slower congruent than incongruent responses (avoidance).

5.3.3. DPT Congruency analysis

Consistent with Koster et al. (2004) emotion congruent and incongruent RTs in TN and PN trials were compared to NN baselines, in a series of ANCOVA using trait anger as a covariate. This allowed componential characteristics to be established. As no significant findings were revealed for TP bias scores, TP word pairs were not examined in the congruency analysis that follows. Degrees of freedom were corrected where appropriate. Descriptive statistics are presented in Table 5.2.

Table 5.2 . Mean RTs in milliseconds (standard deviations in parentheses) for congruent and incongruent TN, PN and NN trials in the RHC, LHC and NHC conditions. Faster emotion congruent than baseline responses reflect vigilance while slower emotion incongruent than baseline responses reflect disengagement difficulties.

Word-pair	Probe position	RHC	LHC	NHC
TN	Congruent	392.86 (46.27)	380.17 (43.23)	393.21 (53.74)
	Incongruent	394.42 (47.27)	391.61 (53.94)	395.28 (51.46)
PN	Congruent	392.72 (45.82)	391.49 (54.79)	392.78 (51.89)
	Incongruent	391.94 (45.62)	390.32 (54.19)	396.82 (52.61)
NN	Baseline	392.87 (52.41)	382.03 (52.95)	396.21 (56.12)

5.3.3.1.Threat word congruency analysis

To explore whether attention bias to threat in the LHC compared to the RHC and NHC conditions reflected vigilance, RTs were entered into a 3 (RHC, LHC, NHC) x 2 (TN congruent, NN) repeated measures ANCOVA. No effect of UHC condition, F (2, 48) = 1.00, p = .38, np^2 = .04, word-pair, F (1, 24) = .228, p = .637, np^2 = .01, and no word-pair x UHC interaction F (1.46, 35.91) = .230, p = .73, np^2 = .01, was observed, suggesting that attentional bias did not reflect vigilance processing.

To ascertain if attentional bias instead reflected threat disengagement difficulty, a 3 (RHC, LHC, NHC) x 2 (TN incongruent, NN baseline) repeated measures ANCOVA was performed on RT data. Results showed no effect of UHC condition, F (2, 48) = .214, p = .81, or word-

pair, F (1, 24) = 1.211, p = .282. However, in support of prediction 3, a significant word-pair x UHC interaction was found, F (2, 48) = 3.22, p < .05, $\eta p2$ =. 20. Contrast showed that in the LHC condition RTs were significantly slower for TN incongruent than NN trials, illustrating difficulty disengaging. No differences between TN incongruent and NN trials were found in the RHC, F (1, 24) = 4.64, p < .05, $\eta p2$ =. 16, r = 0.40 and NHC conditions, F (1, 24) = 6.04, p < .05, $\eta p2$ =. 16. This pattern is shown in Figure 5.2.

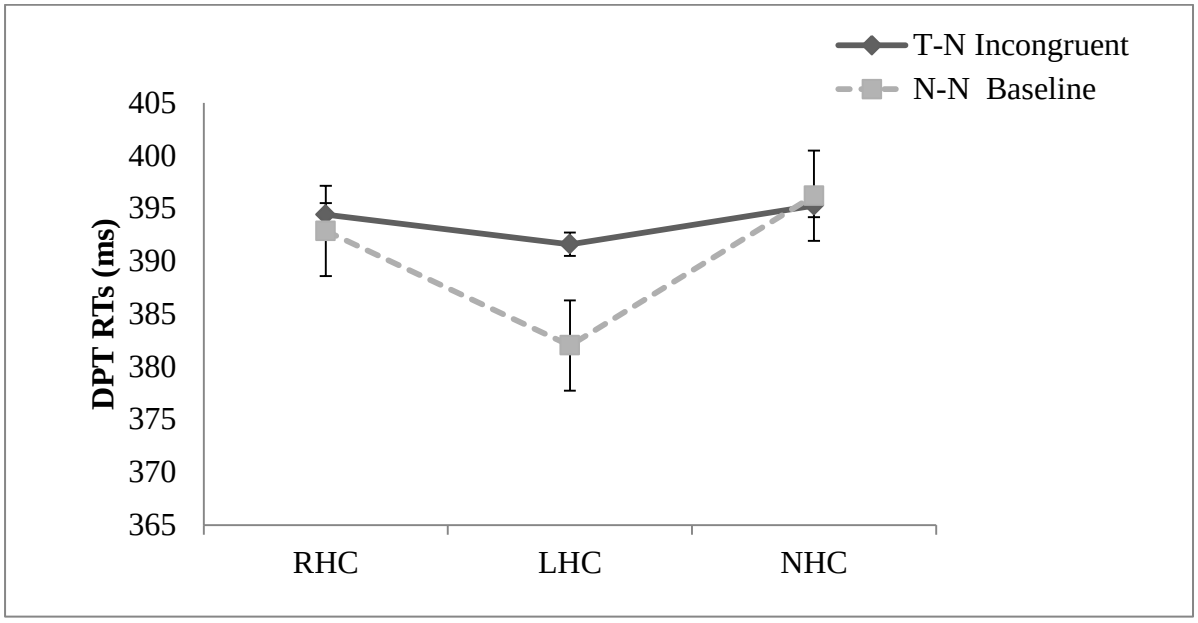


Figure 5.2. Mean DPT RTS for TN incongruent and NN baseline trials in the RHC, LHC and NHC condition with SEM. Slower TN incongruent than NN baseline RTs reflect difficulty disengaging from threat words.

5.3.3.2. Positive word congruency analysis

To explore whether attention bias for positive words in the RHC compared to LHC and NHC reflected vigilance, RTs were entered into a 3 (RHC, LHC, NHC) x 2 (PN congruent, NN) a repeated measures ANCOVA. Results showed no effect of UHC, F (2, 48) = .15, p = .86, np^2

= .01, or word-pair, F (1, 24) = .13, p = .73, np^2 = .01. A significant word-pair x UHC interaction was found, F (2, 48) = 7.01, p < .05, np^2 = .23. Contrasts revealed significantly slower RTs for PN congruent than NN baselines in the LHC condition but faster RTs for PN congruent than NN baseline trials in the NHC condition, F (1, 24) = 9.84, p < .05, np^2 = .29. Contrast also showed no significant RT differences between PN and NN trials in the RHC compared to LHC condition, F (1, 24) = 11.58, p < .05, np^2 = .32 and no RT differences between PN and NN in the RHC compared to NHC condition, F (1, 24) = .025, p > .05, np^2 = .00. This pattern can be seen in figure 5.3.

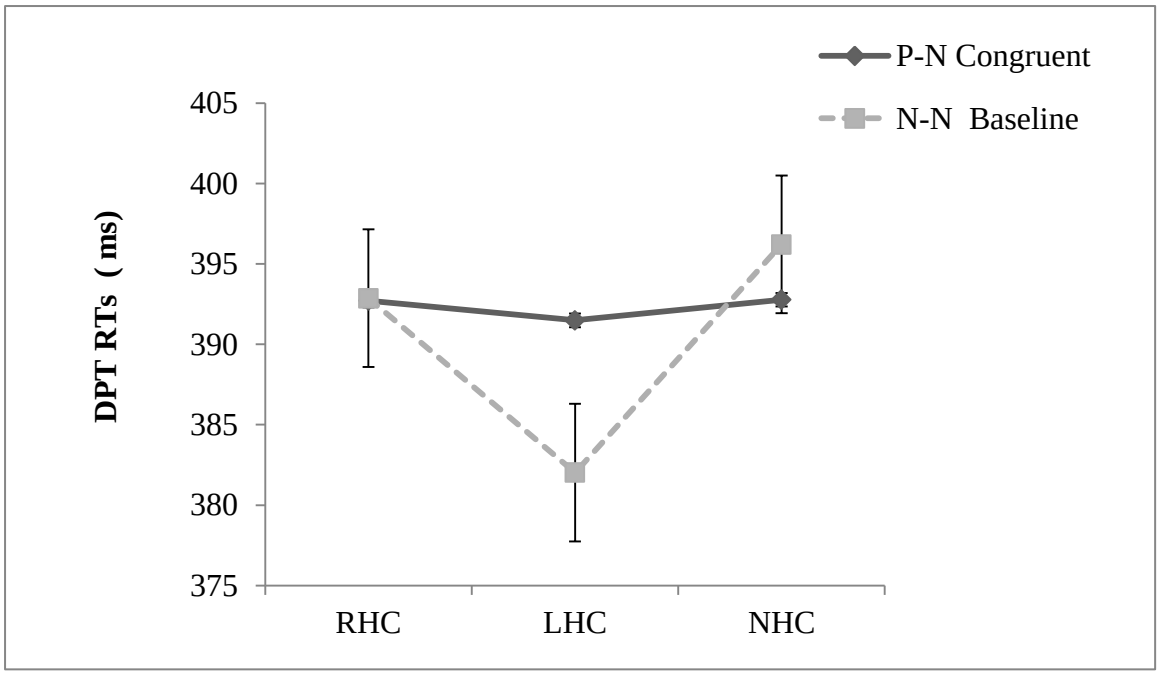


Figure 5.3. Mean DPT RTS for PN congruent and NN baseline trials in the RHC, LHC and NHC condition with SEM. Faster PN congruent than NN baseline RTs reflect vigilance to positive words.

To examine if attentional bias for positive words reflected disengagement difficulties, RTs were entered into a 3 (RHC, LHC, NHC) x 2 (PN incongruent, NN baseline), repeated measures ANCOVA. No effect of UHC, F (2, 48) = .94, p = .34, np^2 = .04, word-pair, F (1, 24) = .39, p = .54, np^2 = .02 or word-pair x UHC interaction, F (2, 48) = 40, p = .67, np^2 = .02, were observed, illustrating no difficulty disengaging from positive words.

5.4. Discussion

The present study explored the effects of the UHC method on modifying components of attentional bias to threat (i.e. vigilance, avoidance and disengagement) using the DPT (MacLeod, et al, 1986; Koster, et al, 2004). This method allowed a detailed exploration of whether the reduced attentional bias induced via RHCs in Studies 1 and 2, was a result of UHCs modulating early vigilance or later disengagement difficulties. Study 3 also aimed to establish whether the reduced attentional biases observed in Studies 1 and 2 reflected greater efficiency of control mechanisms over threat valence or general arousal. In the present study, trait anger was controlled to ensure that any observed effects could be confidently attributed to the UHC and not anger. The findings from this study provide further evidence that UHCs produce attentional changes to emotion related words. Though speculative, these findings may also provide novel evidence that RHCs increased the ability to disengage from threat. Interestingly, the findings also provide novel evidence that LHC increased difficulty disengaging from threat and increased vigilance towards positive words. In doing, so the findings shed light on the conclusions drawn in Study 1 and 2.

In line with prediction 1, there was no difference in DPT response for RHC, LHC or NHC. Based on the assumption that humans show attentional bias to threat (Ohman 2005), it was hypothesised that responses would be faster to probes replacing threat words compared to

probes replacing neutral or positive words. In support of this prediction, results showed an attentional bias towards threat when high arousing threat was presented alongside low arousing neutral words. However, no attentional bias for threat was found when threat words were presented with positive words. Findings also revealed that RTs were slower to threat in TP trials than to positive words in PN trials. This indicated that there was an attentional bias towards positive words regardless of whether they were presented with high arousing threat or low arousing neutral words. The findings of this study also revealed no differences between threat (TN) and positive (PN) responses. Both threat and positive words produced an attentional bias when presented with low arousing neutral words. While these findings supported predictions that independent of UHC, attention would be preferentially oriented to threat over neutral words, the prediction that attention to threat would be prioritised over positive words was not supported. Instead when threat and positive words were presented simultaneously there was an attentional bias towards positive words.

It was also predicted, based on evidence of positive associations between left frontal brain activity and cognitive control (Davidson 2004), that RHCs would reduce threat congruent responses compared to LHCs and NHCs. Results showed reduced attentional bias towards threat in the RHC, indexed by significantly less positive TN responses than in the LHC condition. However, the results also showed that TN responses were significantly less positive in the NHC than the LHC condition and no TN response difference was observed in the RHC and NHC conditions. These findings suggest the congruency effect found in the TN trials was a result of LHC increasing attention bias towards threat rather than RHC reducing it. Therefore, in the present study it is possible that RHCs had no effect on attentional bias to threat and the LHC (compared to NHC) increased attentional bias towards threat.

Exploring the effect of UHCs on positive and neutral words combined showed that PN responses were more negative in the LHC than the NHC condition. However, no differences between RHC and LHC or between RHC and NHC for PN trials were observed. This suggests that LHC (compared to NHC) produced avoidance for positive words. Furthermore, while a pattern of threat avoidance also emerged for TP trials in RHC compared to LHC and NHC, differences were not significant. Nevertheless, it may suggest that when high arousing threat and positive words are presented alongside one another that RHCs might have reduced attentional bias to threat in favour of attending to positive words. This finding is in line with the increased facilitation effect found for RHCs in Study 1 (possible task approach motivation and/or cognitive control) and with the increased neural efficiency of later cognitive control mechanisms for the RHC group in Study 2. This finding also provides novel evidence that RHCs modulated attentional bias to threat valence rather than general arousal.

Furthermore, these findings provides additional support for the view that increased left frontal brain activity increases cognitive control over emotional responding (e.g. Davidson, 2004) and or goal relevant approach motivational processing (Harmon-Jones 2006; Peterson et al. 2008). However, these findings are also consistent with evidence that shows RHCs induce positive affect, bias perceptions and judgments positively (Schiff and Lamon 1994). For example, the avoidance to threat compared to positive words may have reflected attentional bias towards affect congruent positive words. This assumption is in line with evidence that has revealed that trait positive affect is associated with greater left frontal brain activity (Tomarken et al. 1992). However, interpretation from the TP findings should be viewed with caution as the lack of significance makes any assumptions drawn purely speculative. Nevertheless, the findings further highlighted the importance of exploring

underlying neural indices of this effect to gain a better understanding of how the frontal brain is specialised for attentional bias to highly arousing negative and positive stimuli (Chapter 6).

Consistent with Koster et al. (2004), it was also predicted that a reduced attentional bias in the RHC condition would reflect reduced difficulty in disengaging from threat rather than a reduced vigilance, reflecting modulations of later cognitive processes. Indeed, results showed no difference between threat congruent and baseline responses, suggesting the congruency effect did not reflect vigilance for threat. In contrast, significantly slower threat (TN) incongruent than baseline RTs were observed but only in the LHC condition. This illustrated increased difficulty disengaging from threat in the LHC condition, but no disengagement difficulties in RHC or NHC conditions. The prediction that RHC would reduce difficulty disengaging from threat was therefore supported only when comparing RHCs to LHCs in isolation.

Given the lack of difference between RHC and NHC, it was originally inferred that RHCs may have had no effect on attentional bias to threat in the DPT. In contrast, it was inferred that LHCs significantly increased difficulty disengaging from high arousing threat compared to neutral words. This finding was consistent with trends from Study 2 (Chapter 4) that LHCs increased attentional bias to threat and reduced the recruitment of cognitive resources at later inhibitory stages. However, it was also possible that the lack of disengagement difficulties found in the RHC condition reflected an increased ability to disengage which would not be evident from behavioural data alone and would require an exploration of the neural underpinnings of the RHC effect in the DPT (Chapter 6). The lack of difference between RHC and NHC suggesting that the RHC had no effect on attentional bias to threat, illustrates

the importance of including a NHC control in future studies exploring the effects of UHC on modulating attentional bias.

When exploring the componential characteristics of the congruency effects in PN trials, findings revealed faster congruent than baseline RTs in the NHC condition only, suggesting vigilance for positive words when no hand contractions were performed. There were no differences between PN and NN trials in the RHC and LHC condition; rather, both RHC and LHC resulted in avoidance of positive words. This finding illustrated that when positive and neutral words were presented simultaneously, RHCs did not modulate attentional bias to high arousing positive words. This finding mirrored those found for threat versus neutral words suggesting that RHCs produced a similar pattern of findings for highly arousing stimuli rather than for threat per se. In contrast, LHCs produced avoidance for positive compared to neutral words. Therefore, while LHCs increase attentional bias towards threat, they draw attention away from positive words. This finding is in line with evidence that LHCs increase negative affect (Schiff and Lamon 1994) and with evidence that negative affect is positively associated with avoidance of positive stimuli (Roelofs, Putman, Schouten, Lange, Volman, and Rinck 2010). Therefore, these findings provide further evidence that LHCs modulate attentional bias similar to that seen in relation to negative affect.

The findings that LHCs increased difficulty disengaging from threat and reduced vigilance towards positive words are consistent with an association between greater right frontal brain activity and attention bias towards threat and avoidance of positive stimuli (Perez-Edgar et al. 2013). They are also consistent with increased negative affect and the negative biasing of perceptions and judgments following LHCs (Schiff and Lamon 1994). A wealth of evidence

shows a positive association between negative affect and difficulty disengaging from threat (Bar-Haim et al. 2007). However, results showing LHCs produced increased difficulty disengaging from threat conflict with evidence of the association between greater right frontal brain activity and withdrawal from threat (Harmon-Jones et al. 2010). According to the Motivational Model, it would expect that LHCs are associated with avoidance of threat. The findings also contrast with evidence relating greater left frontal activity and attentional bias towards threat (e.g. d'Alfonso et al. 2000) although methodological differences discussed in Chapter 3, may explain this discrepancy.

The findings from the present study suggesting reduced difficulty disengaging from threat for RHCs compared to LHCs are consistent with evidence that RHC affects approach motivation (Harmon-Jones 2006). According to Gray's (1987) Motivational Theory, the behavioural activation system (BAS) motivates and guides movement towards rewarding goals. Therefore, the reduced disengaging difficulty from threat may reflect increased BAS, thereby producing shifts in attention from threat to focus on goal relevant task responses. The findings are also consistent with other cognitive theories (e.g. Davidson 2004). For example, if RHCs reduced difficulty disengaging from threat, this may reflect increased cognitive control over goal directed behaviour (Davidson 2004). Indeed, evidence of increased neural efficiency following RHCs in Study 2 support this view strongly. As such it can be assumed based on findings from Study 1, 2 and 3, increased left frontal activity, via RHCs, may increase the ability to shift attention from task irrelevant information (threat) to assign greater cognitive resources to task relevant responses in the DPT. However, a lack of difference between RHCs and NHCs as well as a lack of neurological insight make these assumptions purely speculative at present.

The findings provide important insights into the effects of UHCs on attentional bias to emotion words. While not fully supporting predictions made, results indicated that LHCs, and presumably right frontal circuits, promote negative affect, difficulty disengaging from threat and avoidance of positive words. In contrast, while speculative, RHCs and presumably left frontal circuits promote increased cognitive control (Davidson 2003; 2004) and/or approach motivation (Harmon-Jones 2006). Uncertainty surrounding the true RHC effects and whether if present it reflects increased cognitive control and/or approach motivation in the DPT needs further examination (Chapter 6).

While the present study provides important insight into the causal role of the left frontal brain in attention bias to threat, no insight has been provided into how trait anger may influence the effect of RHCs. For example, the Integrative Cognitive Model of trait anger (Wilkowski and Robinson 2008) highlights the link between high trait anger and automatic attentional allocation to threat stimuli as well as reduced ability to employ effortful control over dominant ruminative attention. However, as Study 3 did not preselect the sample on high/low trait anger scores Study 3 does not provide insight into how RHCs influences attentional processing in individuals with high compared low trait anger.

Furthermore, while a pattern of avoidance of threat emerged for TP trials in RHCs compared to LHCs and NHCs, differences were not significant. It is important to note that the lack of significance may reflect the fact that trait anger was controlled for rather than investigated alongside the UHC effect. As discussed in previous chapters, the Diathesis Stress Model (e.g. Davidson 1992; Davidson and Tomarken 1989), suggests that increased left frontal brain activity, via RHCs would not be sufficient to induce a change in affective behaviour. Instead, a change in attentional bias to threat would only be expected in response to RHCs in

individuals predisposed to greater left frontal brain activity. Future research should address these issues and preselect groups on trait anger (high, low) (see Chapter 6). Only then, will a real understanding of how transient increases to the left frontal brain influences attentional bias to emotional words.

Furthermore, as only low level, behavioural data was collected in the present study no insight can be provided into the spatial and temporal characterises of underlying neuronal mechanisms related to the UHC effects in relation to components of attentional bias. By converging the DPT paradigm with ERP measures (see Chapter 3 for details on ERP measures), it may be possible to gain insight into the specific underlying mechanisms of the possible reduced difficulty disengaging from threat in the RHC and the increased difficulty disengaging from threat in the LHC condition. ERPs may also provide a means to begin to unravel whether the modulated attentional bias found in the RHC and LHC conditions reflect differing underlying motivational and cognitive control process.

.

The findings of the study presented in this chapter warrant further exploration of how the UHC method modulates attentional bias to threat in the DPT. As questions remain about how the componential characteristics of the UHC effects are influenced by trait anger and how UHC modulate underlying neural indices of attentional disengagement processes, these issues were explored in the next chapter.

CHAPTER 6

STUDY 4: ATTENTIONAL PROCESSING OF EMOTIONAL WORDS: EFFECTS OF LATERALIZED HAND CONTRACTIONS: AN ERP STUDY

6.1. Introduction

The aim of the study presented in this Chapter was to explore which aspects of attention were modulated by UHCs in the previous DPT study (Chapter 5), and how individual differences in trait anger levels interact with UHCs. The lack of research exploring the effects of UHCs on attentional bias warranted a study that could help develop an understanding of the neural mechanisms modulated by this method when used in a DPT paradigm. The research also aimed to explore whether neural changes related to UHCs during the DPT were differentially associated with individual differences in trait anger. As it was assumed in the behavioural DPT study (Chapter 5), that UHCs influenced top-down attentional processing, modulation in later ERP components, namely P2, N2, P3, were explored.

As discussed in Chapter 1, threat-related attentional bias is an underlying mechanism of the fear system that serves a normative function that priorities threat over positive and neutral information allowing rapid detection of, and appropriate responding to threat in the environment (e.g. Öhman 2005). Research has also shown that attentional bias is related to emotional arousal rather than valence (e.g. Kanske and Kotz 2007; Herbert et al. 2008). In order to establish whether attentional bias modifications were a result of attentional changes to valence or arousal, the present study examined the effect of UHCs on attentional bias to high arousing threat and positive words compared to low arousal neutral words.

A non-normative sensitivity to threat, leading to an even more pronounced attentional bias to threat, has been implicated in high anger (e.g. Smith and Waterman 2003; van Honk, Tuiten, de Hann, van den Hout, and Stam 2001). However, as discussed in Chapter 2, EST research offers no insight into underlying components of attentional bias. In contrast, the use of the DPT (MacLeod et al. 1986; Koster et al. 2004) allows a detailed exploration of these attention processing components (see Chapter 2 for details about the EST and DPT method). Through the use of the DPT paradigm, evidence is emerging that attentional bias towards high arousing threat reflects deficits in later cognitive disengagement processes rather than in early preferential vigilance for threat reflecting poor attentional control in relation to negative affect (e.g. Cisler et al. 2009; Derryberry and Reed 2002).

However, as discussed previously, only one DPT study has reported a relationship between high anger and attentional bias towards threat (Smith and Waterman 2003) but analysis to identify whether attentional bias towards threat reflected vigilance or later disengagement difficulties was not reported in that study. Therefore, it still remains unclear which mechanisms of attentional bias are influenced by anger and approach motivation. While evidence is slowly emerging to suggest that attentional biases reflect cognitive impairments it often provides little insight into the underlying neural mechanisms of this effects (Cisler et al. 2009).

Scalp-recorded ERPs, which provide fine-grained temporal information about changes in the order of milliseconds, are particularly sensitive to neural substrates of threat-related attentional processes involved in cue and target stimuli in the DPT. For details about the ERP method see Chapter 2. To date, only a handful of studies have explored ERPs related to approach motivated anger (e.g. Stewart et al. 2010; Bertsch et al. 2009). This research

typically shows that threat-related attentional bias in relation to anger is confined to P2, N2, P3b and NSW components. Details about these components are presented in Chapter 2.

A wealth of ERP research exists that has explored attentional bias in the DPT in relation to anxiety (e.g. Mueller et al. 2009; Helfinstein et al. 2008). However, this has mainly focused on either early occipital generated ERPs (P1, N1) or on the N2pc component, a negative-going deflection at posterior sites to explore visuospatial attentional orientation (e.g. Luck et al. 1990; Hillyard et al. 1995; Fichtenholtz et al. 2007; Fox, Derakshan, and Shoker 2008). To date, only three published anxiety related DPT studies have explored later cognitive ERPs (P2, N2, P3b) during threat processing (Bar-Haim et al. 2005; Eldar and Bar-Haim 2010; Fox et al. 2008) with only one finding a P3b effect (Eldar and Bar-Haim 2010). Eldar and Bar-Haim (2010) showed that reduced attention bias to threat resulted in a reduced P2, increased N2 and reduced P3 amplitudes.

To date, no research has explored ERPs related to anger-related attentional bias using the DPT paradigm. The gap in the literature means that guidance in the current study has been sought from the anxiety literature (Eldar and Bar-Haim 2010) and from literature that provides insight into the relationship between anger and ERP modulations in EST paradigms (Bertsch et al. 2009; Stewart et al. 2010). Previous EST research has highlighted that anger-related attentional bias is indexed by larger P2, N2, and P3 for threat than neutral stimuli (Bertsch et al. 2009; Stewart et al. 2010). This pattern of results suggests that anger-related attentional bias is related to increased allocation of attentional resources to evaluate threat and the exertion of more motivational and inhibitory resources to suppress attentional bias towards threat. As previous ERP studies exploring attentional bias in high anger employed the use of the EST, the findings provide no insight into the underlying mechanisms of

attentional components. Furthermore, the correlational nature of these studies means that it remains unclear how individual differences in anger and approach motivation are causally related to attentional bias to threat.

In previous chapters the UHC method (Schiff and Lamon 1994) has been shown to modulate attentional bias. As such the UHC method provides a possible means to explore any causal relationship between induced increases in approach motivated and anger-related brain networks (Harmon-Jones 2006; Peterson et al. 2008) and attentional bias to threat. However, there has been no research exploring whether UHCs will modulate the neural indices of attentional bias to emotion related words in the DPT. The aim of Study 4 was to explore whether the increased difficulty disengaging from threat found in the LHC compared to the RHC condition in Study 3 (Chapter 5), was related to increased threat evaluation, increased motivation towards threat, and/or reduced cognitive control. The present study also aimed to provide insight into whether RHCs compared to LHCs and NHCs reduced difficulty disengaging from threat as this remained unclear from Study 3. Exploring the effects of RHCs and LHCs compared to NHCs on ERP components related to evaluative (P2), early cognitive control (N2) and motivational (P3b) aspects of attentional bias to emotional words may provide some clarity into these questions. The exploration of the NSW component was not examined in the present study as recent evidence suggests that this component is typically related only to EST interference (van Hooff, et al. 2008).

6.1.1. Predictions

6.1.1.1. Behavioural element

As Study 3 did not explore the effects of UHC in relation to individual differences in anger, predictions were based on guidance from the literature. While exploratory in nature clear

predictions were made in the present study. These were based on evidence that attentional bias is more evident to threat over positive and neutral stimuli (e.g. Öhman 2005). Predictions are also based on findings from Study 1, 2 and 3 that showed that RHCs reduced attentional bias to threat, increased the efficiency of later cognitive control mechanisms and increased the ability to disengage from threat respectively. The predictions are also based on evidence that the left frontal brain is involved in cognitive control over emotional responding (e.g. Davidson 2004), that high anger increases attentional bias towards threat (e.g. Smith and Waterman 2003) and that attentional bias to threat reflects disengagement processes rather than vigilance (e.g. Cisler et al. 2009). In line with this evidence the following predictions were made:

1. There will be no main effect of UHC or trait anger on DPT bias-scores.
2. There will be increased attentional bias for threat compared to positive and neutral words. This will be indexed by more positive bias-scores (reflecting faster RTs to threat congruent than incongruent targets) for TN and TP compared to PN word-pairs.
3. RHCs (relative to LHCs and NHCs) will reduce attentional bias to threat compared to positive and neutral words. This will be indexed by reduced positive TN bias-scores in the RHC relative to LHCs and NHCs, (reflecting similar RTs for congruent and incongruent targets in TN and TP compared to PN trials) and resulting in equal bias-scores for TN, TP and PN trials.
4. High compared to low anger participants, will show increased attentional bias to threat compared to positive and neutral words. This will be indexed by more positive bias-scores (reflecting faster RTs to congruent than incongruent targets) for TN and TP compared to PN word-pairs in high compared to low anger participants.

5. RHCs (compared to LHCs and NHCs) will reduce attentional bias to threat (compared to positive and neutral words) and this effect will be larger in high than low anger participants. This will be indexed by larger effects of reduced positive bias-scores in TN and TP compared to PN trials for high relative to low anger participants in the RHC (reduced positive TN and TP bias-scores reflect smaller RTs difference for congruent and incongruent targets).
6. The reduced attentional bias to threat versus positive and neutral words for RHCs compared to LHCs and NHCs will reflect reduced disengagement difficulty rather than reduced vigilance. This will be indexed by slower threat incongruent than baseline RTs.

6.1.1.2. ERP element

Given the lack of anger literature guidance was sought from the anxiety literature which has shown reduced attentional bias towards threat, is associated with reduced P2 and P3 amplitudes and increased N2 amplitudes in the DPT (Eldar and Bar-Haim 2010). It was also assumed that if observed behavioural biases were a result of later cognitive processing of high arousing emotional cues that the effects of UHC would be reflected in ERP components time locked to DPT target onset. The ERP predictions were also based on the findings from Studies 1, 2 and 3 that showed RHCs reduced attentional bias to threat, increased later cognitive control and increased ability to disengage attention from threat. They are also based on evidence that high anger is associated with greater left frontal brain asymmetry (e.g. Harmon-Jones 2004) and attentional bias towards threat (Smith and Waterman 2003). Predictions regarding laterality were also made as findings supporting these may provide evidence of the viability of UHCs to induce contralateral brain activity changes. Based on the novelty of Study 4 and corresponding paucity of previous literature, higher order predations

are more speculative and would be based on experimental controls. For these reasons, the following predictions were made:

7. There will be no main effect of UHC, anger, congruency, hemisphere or region on the P2, N2 and P3b.
8. In high compared to low anger participants, P2 and P3b amplitudes will be larger and N2 will be smaller for threat (compared to positive or neutral) congruent than incongruent targets.
9. In the RHC compared to the LHC and NHC conditions, P2 and P3b will be smaller and N2 will be larger for threat (compared to positive or neutral) congruent than incongruent targets.
10. In the RHC compared to the LHC and NHC conditions, P2 and P3b will be smaller and N2 will be larger for threat (compared to positive or neutral congruent) than incongruent targets and this effect will be larger in high than low anger participants.
11. In high compared to low anger participants, P2, N2, and P3b will be larger in the left than the right hemisphere.
12. In the RHC compared to the LHC and NHC conditions, P2, N2, and P3b will also be larger in the left than the right hemisphere.

6.2. Method

6.2.1. Design

6.2.1.1. Behavioural element

To examine the effects of UHCs on attentional bias to emotion-related words in relation to trait anger the study employed a 3 (UHC condition; RHC, LHC, NHC) x 4 (word-pair; threat-neutral (TN), threat-positive (TP), positive-neutral (PN), neutral-neutral (NN) x 2 (probe

position; congruent, incongruent) x 2 (trait anger group; high; low) multifactorial mixed design. For first phase of the behavioural element of the study that aimed to explore the effects of UHCs on DPT measured attentional bias to emotional words the dependent variable was DPT bias scores. It is important to note that the use of bias scores as a DV eliminates the factor of congruency as well as the need for NN trials as congruence cannot be differentiated in these trials (see Section 2.6.1. for detail about bias-score production). In the second phase of the behavioural element of the study that aimed to explore whether any attentional bias to emotional words reflected vigilance or disengagement difficulties the dependent variable was DPT RTs (ms). This exploration included the factor of congruency and required the use of NN trials (see Section 2.6.2. for detail about congruency analysis).

6.2.1.2. ERP Element

6.2.1.2.1. P2 Component

To examine the effects of UHCs on neural indices vigilance to emotion-related words in relation to trait anger the study employed a 3 (UHC condition; RHC, LHC, NHC) x 3 (word-pair type; threat-neutral (TN), threat-positive (TP), positive-neutral (PN) x 2 (probe position; congruent, incongruent) x 2 (trait anger group; high; low) x 2 (Region, frontal, frontocentral) x 2 (laterality, left, right) multifactorial mixed method design. The dependent variable was the attention related ERP component P2 in response to DPT targets.

6.2.1.2.2. N2 Component

To examine the effects of UHCs on neural indices of cognitive control over attentional bias to emotion-related words in relation to trait anger the study employed a 3 (UHC condition; RHC, LHC, NHC) x 3 (word-pair type; threat-neutral (TN), threat-positive (TP), positive-neutral (PN) x 2 (probe position; congruent, incongruent) x 2 (trait anger group; high; low) x

3 (Region, frontal, frontocentral, central) x 2 (laterality, left, right) multifactorial mixed method design. The dependent variable was the attention related ERP component N2 in response to DPT targets.

6.2.1.2.3. P3b Component

To examine the effects of UHCs on neural indices of motivational factors of attentional bias to emotion-related words in relation to trait anger the study employed a 3 (UHC condition; RHC, LHC, NHC) x 3 (word-pair type; threat-neutral (TN), threat-positive (TP), positive-neutral (PN) x 2 (probe position; congruent, incongruent) x 2 (trait anger group; high; low) x 3 (region, centroparietal, parietal, parietooccipital) x 2 (laterality, left, right) multifactorial mixed method design. The dependent variable was the attention related ERP component P3b in response to DPT targets.

6.2.2. Participants

Participants were 44 undergraduate students who scored in the top or bottom 30% of the AX-O subscale of the STAXI-2 (Spielberger 1999) in the sampling phase of the study. For information on this first phase and the STAXI-2, see Sections 2.2 and 2.3.2 respectively.

24 participants were classified as high anger (AX-O Score; *M* = 20.38, *SD* = 2.94), 16 of which were female (aged; *M* = 19.13, *SD* = 2.03) and 8 male (aged; *M* = 19.37, *SD* = 1.06). 20 participants were classified as low anger (AX-O Score; *M* = 12.05, *SD* = 1.76), 13 of which were female (aged; *M* = 20.23, *SD* = 3.94) and 7 male (aged; *M* = 20.14, *SD* = 2.79). Participants also met the inclusion criteria set out in Chapter 2. Participants received course credits for their participation. Coventry University Ethics Committee approved the study and

ethical guidelines were adhered to. Details about the inclusion criteria and ethics are presented in Section 2.2.

6.2.3. Materials

6.2.3.1. DPT word stimuli

Information about the word stimuli for this study can be found in Section, 2.5.4. The list of words used in Study 4 is also presented in Appendix 2.

6.2.3.2. The Dot-Probe Task (DPT)

Information about the DPT experimental protocol used in this and the next study are presented in detail in Section 2.5.3.

6.2.4. The UHC method

Information about the UHC procedure is presented in detail in Section 2.4.

6.2.5. Procedure

Before the DPT, participants were given a participant information sheet, provided informed consent and were prepared for EEG recordings. Participants completed the DPT in all UHC conditions in a single session. Participants completed hand contractions immediately prior to task onset. Latin squared counterbalancing dictated which UHC condition (RHC, LHC, or NHC) was performed before each DPT block onset. Participants were prepared for EEG (see Chapter 2 for information about this procedure) and standardised verbal instructions on how to complete the designated hand contraction were provided. During the DPT participants

responded as quickly as possible to the probe location through a button press. Each session lasted 70 minutes, with 5-minute breaks between blocks to reduce fatigue effects.

6.2.6. EEG Acquisition

Information regarding data acquisition procedures is presented in Section 2.7.2.

6.2.7. Data Reduction and Statistical Analysis

6.2.7.1. Preparation of Behavioural Data

All trials where participants had either pressed the wrong key or missed a response entirely were rejected as error (M = 1.54 SD = 3.03). Then premature and extremely long reaction times faster than 200 ms or slower than 1500 ms were removed (M= .18 %, SD =.38). RTs more than three STD from each individuals mean were then removed (M= 3.80 %, SD = .82). Overall, mean percentages of data lost as either an error or an outlier was 5.5 %, SD = 3.44.

An alpha level of 0.05 was used for all statistical tests. All analyses were performed two-tailed.

6.2.7.2. Preparation of EEG-Data

Continuous EEG raw data was processed according to the procedure set out in section 2.8. As discussed in Section 2.8, separate grand average ERPs were computed for each condition; T-N congruent, T-N incongruent, P-N congruent, P-N incongruent, T-P congruent, and T-P incongruent. Electrode positions and scoring epochs P2 (190-260 ms), N2b (250-310 ms), and P3b (290-550 ms) were selected following visual inspections of grand ERP waveforms and referring to previous DPT ERP literature (e.g. Bar-haim et al, 2005; Eldar and Bar-Haim, 2010; O'Toole and Dennis, 2012;). The P2 was scored at frontal (F3 and F4) and

frontocentral (FC1 and FC2) electrode sites. The N2b was scored at frontal (F3 and F4), frontocentral (FC1 and FC2), and central (C3and C4) electrode sites. The P3b was scored at centroparietal (CP1 and CP2), parietal (P3 and P4), and parietooccipital (PO3 and PO4) sites.

6.3. Results

For the sake of brevity only significant inferential statistics which directly correspond to the main predictions of the present study (outlined in section 6.1.1) will be presented in detail. All other results are presented in table format.

6.3.1. Behavioural Element

To explore the effect of UHC condition on attentional bias to high arousal emotion words in relation to anger, DPT bias-scores were entered into a 3 repeated measures (UHC; RHC, LHC, NHC) x 2 (Word-pair; TN; PN, TP) x 2 between subject (trait anger group; high, low) mixed design ANOVA. Significant interactions that included only within subject factors were explored using repeated measure contrasts and interactions that included both within and between subject factors were explored using one-way ANOVA and paired t-tests. Bonferroni corrections were applied for multiple comparisons. Data met ANOVA assumptions. Mauchly's test indicated that sphericity was assumed for the main effects of UHC, χ^2 (2) = 4.39, $p > .05$ and word-pair, χ^2 (2) = 1.46, $p > .05$ but was violated for the UHC X word-pair interaction, χ^2 (9) = 20.86, $p < 0.05$. Therefore, degrees of freedom were corrected ($\varepsilon = .91$) using Greenhouse-Geisser estimates of sphericity for the UHC x word-pair interaction.

Descriptive statistics for each condition for high and low anger participants are shown in Table 6.1 and inferential statistics of this analysis are presented in Table 6.2 and for brevity

only significant findings directly relating to predictions are discussed in detail other findings are represented in table format..

Table 6.1. Mean DPT bias-scores with standard deviation in parenthesis for TN, PN and TP word-pairs for the RHC, LHC and NHC conditions in high and low anger participants

Anger group	**Word-pair**	**UHC**	***M***	***(SD)***
High anger (*N* 24)	TN	RHC	-2.18	(14.39)
		LHC	2.43	(12.50)
		NHC	-2.45	(17.02)
	PN	RHC	-2.65	(10.81)
		LHC	2.71	(14.33)
		NHC	-1.42	(14.27)
	TP	RHC	3.99	(10.20)
		LHC	-2.84	(15.34)
		NHC	-0.30	(6.27)
low anger (*N* 20)	TN	RHC	-4.44	(6.38)
		LHC	-9.24	(17.53)
		NHC	-5.30	(16.66)
	PN	RHC	-2.16	(12.76)
		LHC	-5.04	(12.94)
		NHC	-5.36	(13.83)
	TP	RHC	-0.95	(8.38)
		LHC	4.38	(13.83)
		NHC	5.25	(11.67)

Table 6.2. Inferential statistics for the effects of UHC condition, trait anger, and word-pair, on DPT bias-scores. Significant effects and interactions are highlighted.

Condition	***F***	***P***
Trait anger	2.55	.12
UHC	0.02	0.98
UHC × trait anger	0.55	0.58
Word-pair	5.67	0.01
Word-pair × trait anger	3.67	0.03
UHC × Word-pair	0.28	0.86
UHC × Word-pair × trait anger	2.72	0.04

As expected (prediction 1), there was no main effect of UHCs or trait anger on DPT responses (p> .05, See Table 6.2 for inferential results). In line with prediction 2, the ANOVA revealed a significant main effect of word-pair, F (2, 78) = 5.67, P < 0.05, np^2 = .12. However, contrary to predictions that there would be an attentional bias for threat compared to positive and neutral words, contrasts revealed that, TN bias-scores were significantly more negative than TP bias-scores F (1, 42) = 9.10, p < .05, np^2 = .18. PN bias scores were also significantly more negative than TP bias scores, F (1, 42) = 5.85, p < .05, np^2 = .12. So while there was an attentional bias towards threat in TP trials there was an avoidance of threat and positive words (in TN and PN trials) with no difference between TN and PN bias-scores, F (1, 42) = .71 p > .05, $np2$ = .02.

Contrary to prediction 3, that compared to LHCs and NHCs, RHCs would reduce attentional bias to threat compared to positive and neutral words, the ANOVA revealed no difference in bias scores for TN, PN and TP word-pairs in relation to the UHC conditions, F (3.25, 136.64) = .28, p > 0.05, np^2 = .01.

In partial support of prediction 4, the ANOVA revealed a significant word-pair × anger interaction, F (2, 78) = 3.67, $P < 0.05$, np^2 = .08. Post hoc tests revealed no difference between high and low anger participants for PN bias scores F (1, 42) = 1.25, $p > 0.05$ or TP bias scores F (1, 42) = 2.78, $P > 0.05$. Both high and low anger participants showed avoidance of positive words (in PN trials) and vigilance towards threat (in TP trials). High and low anger participants were found to differ in response to TN trials. However, contrary to predictions, high anger participants had significantly less negative TN bias scores than low anger participants, F (1, 42) = 5.69, $p < 0.05$. This indicated that there was no attentional bias to threat present in high anger participants but that low anger participants showed avoidance for threat.

With respect to prediction 5, a significant UHC × word-pair × anger interaction was observed, F (3.25, 136.64) = 2.72, $p < 0.05$, np^2 = .06. The interaction effect is presented in Figures 6.1 and 6.2. Further analysis revealed that there was no difference between high and low anger participants for PN word-pairs in the RHC, F (1, 42) = .02, $P > 0.05$, LHC, F (1, 42) = 3.48, $P > 0.05$, or NHC condition, F (1, 42) = .86, $P > 0.05$. Similarly, there was no difference between high and low anger participants for TP word pairs in the RHC, F (1, 42) = 3.00, $P > 0.05$, the LHC, F (1, 42) = 2.64, $P > 0.05$ or the NHC conditions, F (1, 42) = 4.05, $P > 0.05$. It was predicted that, RHCs compared to LHCs and NHCs, would reduce attentional bias to threat compared to positive and neutral words and this effect would be larger in high than low anger participants (hypothesis 5) . This prediction was not supported. There was no difference between high and low anger participants for TN word pairs, in the RHC, F (1, 42) = 4.20, $P > 0.05$ or NHC F (1, 42) = .31, $P > 0.05$ conditions. Interestingly, analysis revealed a significant difference between high and low anger participants for TN word-pairs, in the LHC condition, F (1, 42) = 6.61, $P > 0.05$. In the LHC condition, high

anger participants showed attentional bias towards threat but low anger participants showed threat avoidance. This pattern can be seen in Figure 6.2.

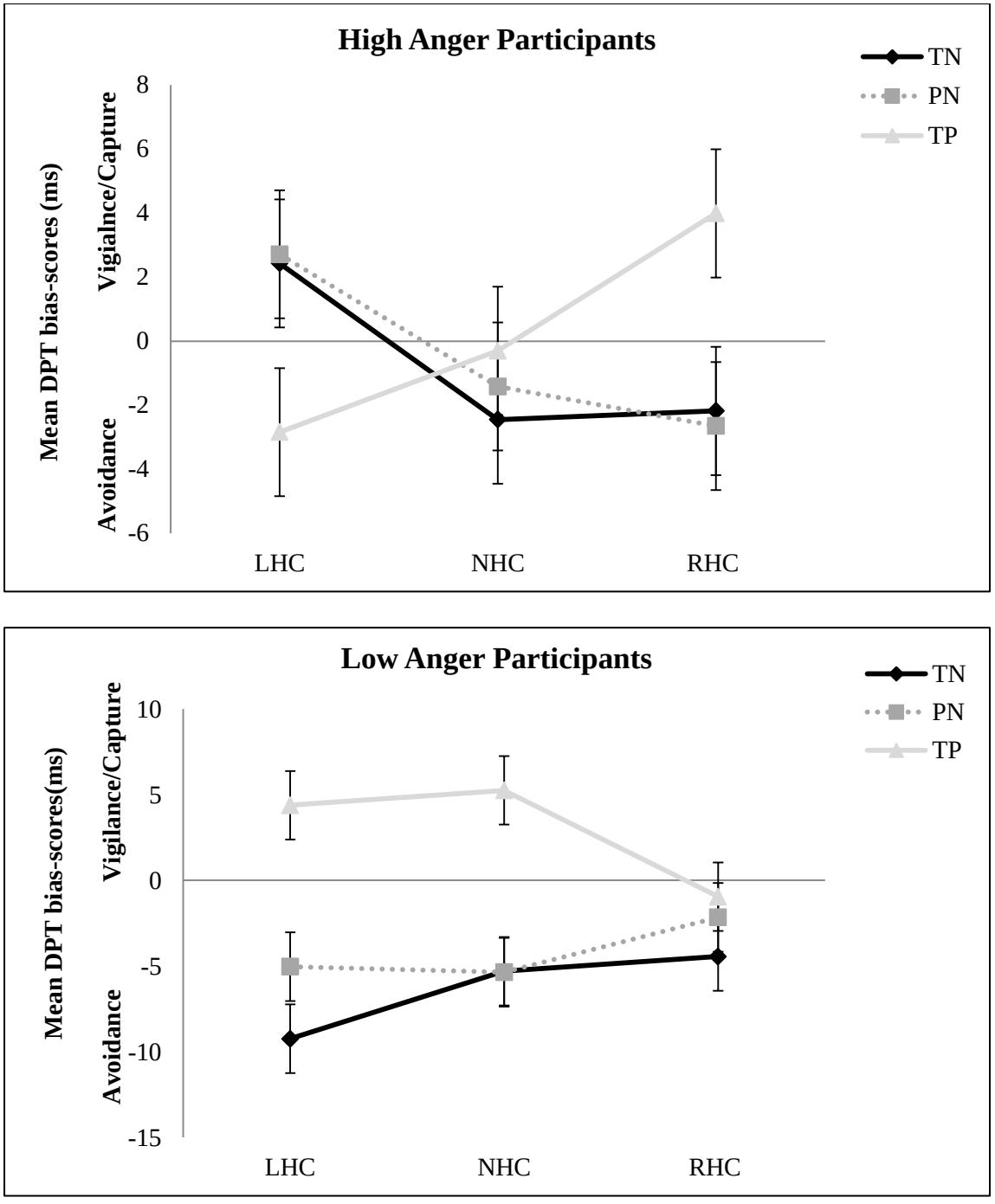


Figure 6.1. Mean DPT bias-scores (mean RT to locate congruent target minus mean RT to locate incongruent targets) in high anger (upper panel) and low anger (lower panel) participants, for TN, PN and TP word-pairs, in LHC, NHC and RHC conditions with SEM. NN trials are not included, as congruence cannot be differentiated. Positive bias-scores reflect faster congruent than incongruent RTs (vigilance/disengagement difficulties). Negative bias-scores reflect slower congruent than incongruent RTs (avoidance).

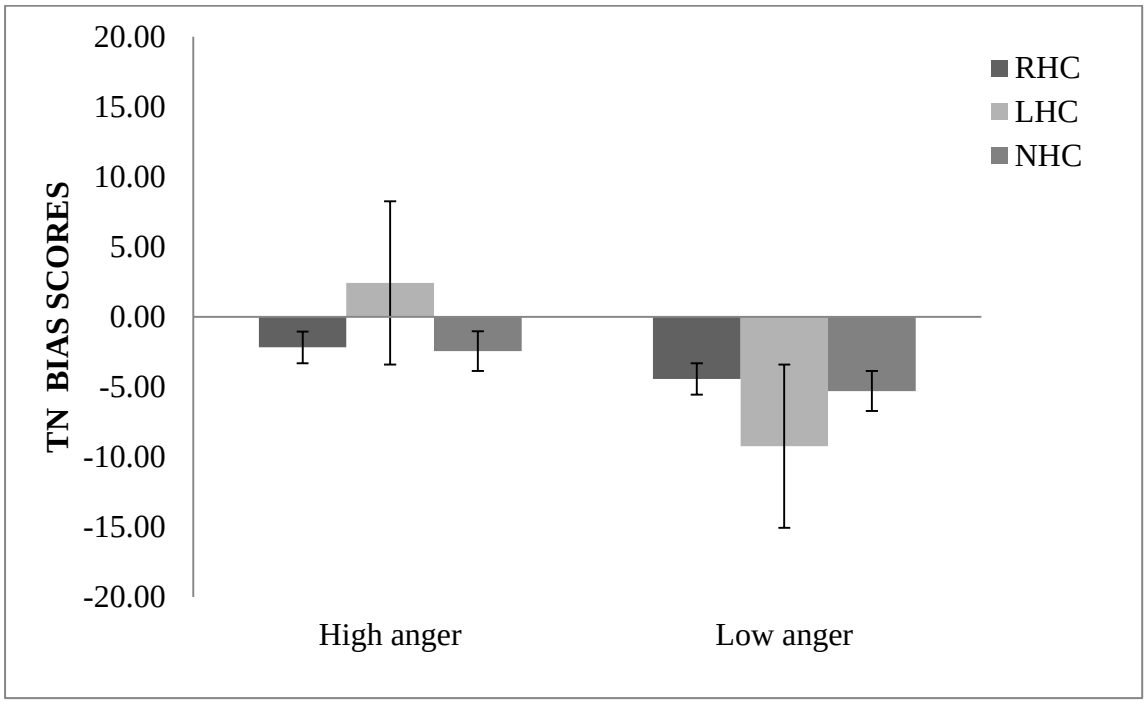


Figure 6.2. High and low anger participants mean DPT bias-scores for TN word-pairs in the RHC, LHC and NHC conditions with SEM. NN trials are not included, as congruency cannot be differentiated. Positive bias-scores reflect faster congruent than incongruent responses (vigilance/disengagement difficulties). Negative bias-scores reflect slower congruent than incongruent responses (avoidance).

Given that in high anger participants RHC resulted in negative bias-scores (avoidance or reduced disengagement difficulties) and LHCs resulted in positive bias-scores (vigilance or disengagement difficulties) it was important to address prediction 6, that a congruency analysis was performed (Koster et al, 2004). The componential characteristics of attentional bias toward threat was explored in a series of paired samples t-tests comparing threat congruent or incongruent RTs with NN baselines. To explore whether the positive TN bias-scores following LHC in high anger participants reflected vigilance, TN congruent and

baseline RTs were compared. TN congruent RTs (*M* = 398.95, *SD* = 68.13) were slower than baseline RTs (*M*= 393.10, *SD* = 63.39), but this difference was not significant, *t* (23) = .57, p = >.05, showing no vigilance for threat. To identify instead whether this attentional bias reflected disengagement difficulties, TN incongruent and baseline RTs were compared. TN incongruent RTs (*M*= 401.38, *SD* = 69.78) were slower than baseline RTs (*M*= 393.10, *SD* = 63.39) but this difference was not significant, *t* (23) = .80, p = >.05 showing no difficulty disengaging form threat. Therefore, it can be assumed that the positive bias-scores for TN word-pairs were not large enough to be classified as true attentional bias towards threat.

6.3.2. ERP Element

For interpretational ease, P2, N2b and P3b ERP amplitude data were subjected to a series of separate 4-way mixed factorial ANOVAs that compared effects of UHC, congruency, region and hemisphere for each of the word-pairs (TN, PN, TP) separately. Significant interactions that included only within subject factors were explored using repeated measure contrasts and interactions that included both within and between subject factors were explored using one-way ANOVA and paired t-tests. Bonferroni corrections were applied for multiple comparisons. As discussed in depth in Chapter 2, type 1 errors were further protected by applying Greenhouse-Geisser epsilon adjustment for all ERP results presented in this chapter.

6.3.2.1. TN word-pairs

6.3.2.1.1. P2 ERP component

To explore whether RHCs compared to LHCs and NHCs reduced P2 (reduced attentional bias towards threat) for highly arousing threat words when presented alongside low arousal neutral words in high compared to low anger participants, a 3 (RHC, LHC, NHC) x 2 (Trait anger group; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left,

right) X 3 (Region; frontal, frontocentral) mixed ANOVA was performed on P2 peak amplitudes to TN targets. Descriptive statistics of P2 amplitudes to TN targets in response to the UHC, congruency, and anger group at left and right frontal and frontocentral sites are presented in Table 6.3. Inferential statistics for this analysis are presented in Table 6.4.

Table 6.2. Means and standard deviations of P2 amplitudes (μV) for TN targets in relation to the UHC, congruency, and trait anger at left and right frontal and frontocentral sites

UHC	**TN congruence**	**Site**	**High Anger (= 24)**		**Low anger (n=20)**	
			M	*(SD)*	*M*	*(SD)*
RHC	TN congruent	F3	0.18	(1.25)	0.50	(1.61)
		F4	0.95	(1.75)	0.57	(1.27)
		FC1	0.53	(1.55)	0.79	(1.59)
		FC2	1.02	(1.36)	0.87	(1.62)
	TN incongruent	F3	0.27	(1.29)	0.83	(1.33)
		F4	0.57	(1.67)	0.85	(1.07)
		FC1	0.72	(1.31)	1.00	(1.86)
		FC2	1.09	(1.46)	0.96	(1.66)
LHC	TN congruent	F3	0.32	(1.21)	0.33	(1.66)
		F4	0.71	(1.30)	0.65	(1.42)
		FC1	0.85	(1.10)	0.88	(1.45)
		FC2	1.04	(1.54)	1.02	(1.33)
	TN incongruent	F3	-0.02	(1.05)	0.83	(1.58)
		F4	0.25	(1.05)	-0.01	(1.35)
		FC1	0.28	(1.43)	0.71	(1.91)
		FC2	0.81	(0.98)	0.40	(1.91)
NHC	TN congruent	F3	0.16	(1.18)	-0.04	(1.44)
		F4	0.51	(0.97)	0.29	(1.69)
		FC1	0.67	(0.87)	0.82	(1.33)
		FC2	0.86	(1.12)	0.89	(1.81)
	TN incongruent	F3	-0.01	(1.39)	-0.13	(1.58)
		F4	0.39	(1.25)	0.42	(1.66)
		FC1	0.78	(1.03)	1.09	(1.97)
		FC2	1.03	(1.28)	1.27	(2.02)

Table 6.4. Inferential statistics for the effect of UHC, congruence, region and hemisphere on P2 peak amplitudes (μV) to TN targets. Significant effects are highlighted.

	F	***p***
Trait anger	.09	.76
UHC	.80	.45
UHC × Trait anger	.05	.94
Congruency	.29	.60
Congruency × Trait anger	1.43	.23
Site	13.86	.001
Site × Trait anger	.01	.94
Hemisphere	3.14	.051
Hemisphere × Trait anger	2.42	.12
UHC × Congruency	.87	.42
UHC × Congruency × Trait anger	.02	.98
UHC × Site	5.04	.010
UHC × Site × Trait anger	1.43	.25
Congruency × Site	.38	.54
Congruency × Site × Trait anger	1.39	.24
UHC × Congruency × Site	1.82	.17
UHC × Congruency × Site × Trait anger	.48	.62
UHC × Hemisphere	.83	.44
UHC × Hemisphere × Trait anger	1.33	.27
Congruency × Hemisphere	1.52	.22
Congruency × Hemisphere × Trait anger	.74	.40
UHC × Congruency × Hemisphere	1.14	.32
UHC × Congruency × Hemisphere × Trait anger	2.07	.13
Site × Hemisphere	.32	.57
Site × Hemisphere × Trait anger	.01	.93
UHC × Site × Hemisphere	.80	.45
UHC × Site × Hemisphere × Trait anger	.19	.82
Congruency × Site × Hemisphere	2.97	.09
Congruency × Site × Hemisphere × Trait anger	.11	.75
UHC × Congruency × Site × Hemisphere	1.39	.26
UHC × Congruency × Site × Hemisphere × Trait anger	.36	.69

In support of prediction 7, the ANOVA revealed no main effect of UHC, anger, congruency, or hemisphere on P2 in response to TN targets (p >.05). Inferential information for these results can be found in Table 6.4. However, an effect of region was revealed, with significantly larger P2 amplitudes at frontocentral than frontal regions, F (1.00, 42.00) = 13.86, P < 0.01, np^2 = .25. This was qualified by a significant UHC × region interaction, F (1.85, 77.99) = 5.04, p <.05, np^2 =.10. Planned contrast showed that P2 amplitude was larger for RHCs than NHCs at frontal sites but did not differ for RHC and NHC at frontocentral sites, F (1, 42) = 9.32, p <.05, np^2 =.18. Contrasts also showed larger P2 amplitudes for LHC than NHC at frontal sites, but smaller P2 amplitudes for LHC than NHC at frontocentral sites, F (1, 42) = 7.49, p <.05, np^2 =.15. Contrasts also showed no significant P2 UHC amplitude difference between frontal and frontocentral sites, P2 amplitudes were larger for RHC than LHC at both, F (1, 42) = .26, p >.05, np^2 =.01. This can be seen in Figures 6.3 and 6.4.

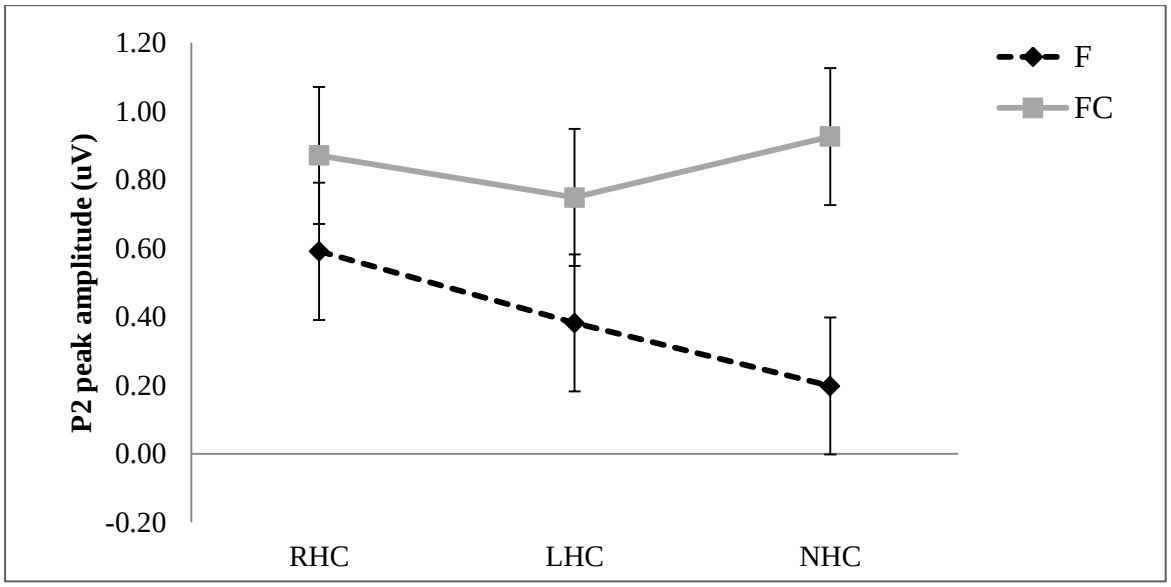


Figure 6.3 Frontal P2 peak amplitudes (μV) for RHCs, LHCs and NHCs for TN DPT trials Data are expressed as mean (SEM)

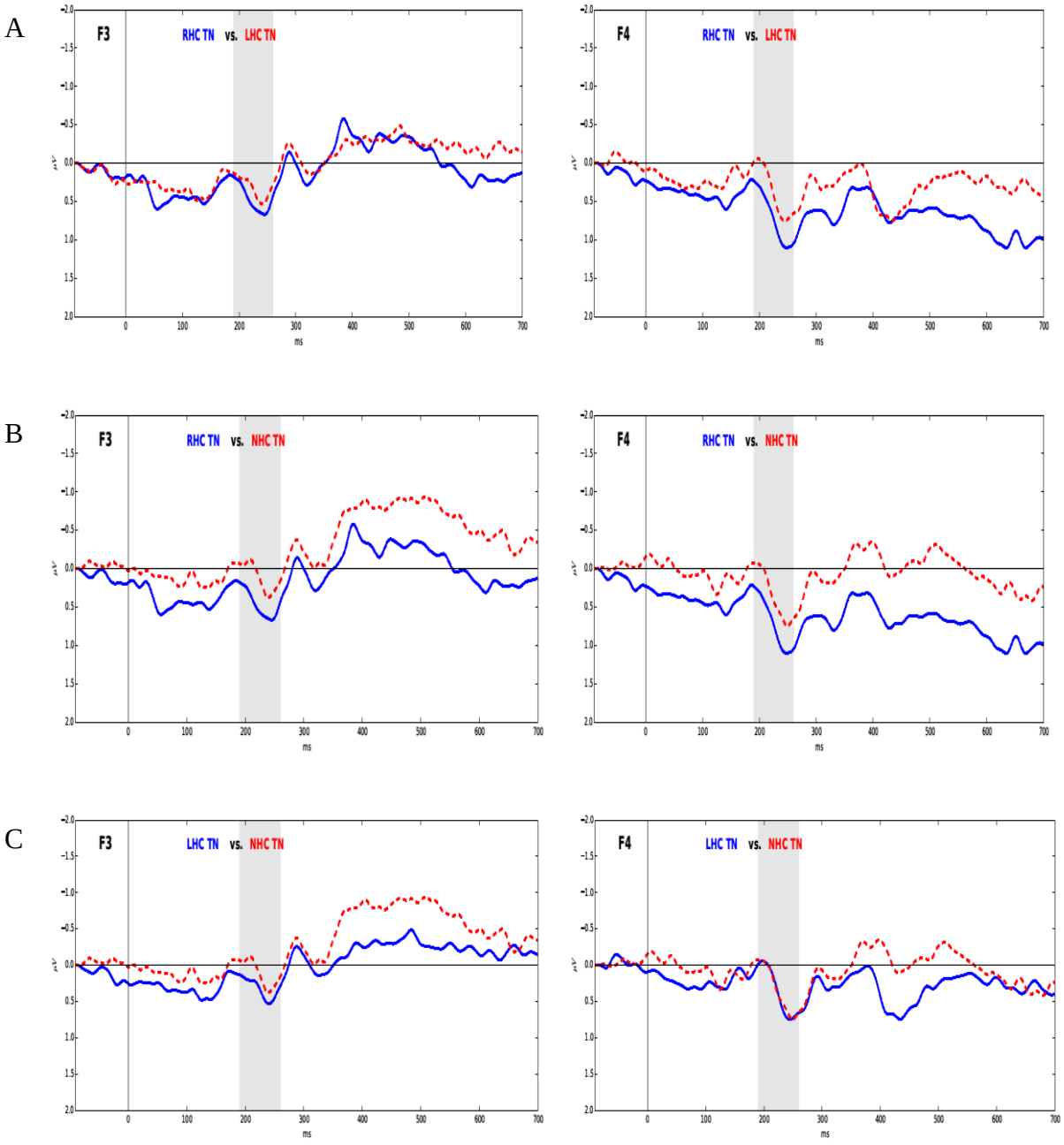


Figure 6.4 Grand average waveforms showing frontal P2 μV (shaded grey) for RHCs vs. LHCs (panel A), RHCs vs. NHCs (panel B) and LHCs vs NHCs (panel C) at F3 and F4 in response to TN trials in the DPT

Contrary to predictions 8 and 9, no P2 difference was found between high and low anger participants, or between RHC compared to LHC and NHC, for TN congruent than incongruent targets ($p > .05$). The ANOVA also revealed contrary to prediction 10, no P2 difference for RHC compared to LHC and NHC, for TN targets in relation to trait anger ($p > .05$). Furthermore, contrary to predictions 11 and 12 regarding the laterality of UHCs and anger, no P2 laterality difference was found between high compared to low anger participants, or between RHC, LHC, and NHC for TN targets ($p > .05$). Inferential statistics for these results can be found in Table 6.4.

6.3.2.1.2. N2 ERP component

To explore whether compared to LHCs and NHCs, RHCs increased N2 (reduced attentional bias towards threat) for congruent compared to incongruent TN word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (Trait anger; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) x 3 (Region; frontal, frontocentral, central) mixed ANOVA was performed on N2 peak amplitudes in response to TN targets. Descriptive statistics for N2 amplitudes in response to UHC, TN congruency and trait anger at left and right frontal, frontocentral and central sites are presented in Table 6.5. Inferential statistics for this analysis are presented in Table 6.6.

Table 6.5. Means and standard deviations of N2 amplitudes (μV) for TN targets in relation to the UHCs, congruency, and anger at left and right frontal, frontocentral and central sites

UHC	TN congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD)*	*M*	*(SD)*
RHC	TN congruent	F3	-0.48	(1.47)	-0.50	(2.15)
		F4	0.75	(2.14)	0.17	(1.390
		FC1	0.22	(1.61)	0.38	(1.74)
		FC2	1.12	(1.78)	0.59	(1.86)
		C3	-0.76	(1.15)	0.13	(1.71)
		C4	0.55	(1.48)	0.71	(1.39)
	TN incongruent	F3	-0.92	(1.59)	-0.21	(1.73)
		F4	0.18	(2.23)	0.63	(1.23)
		FC1	-0.01	(1.97)	0.80	(2.05)
		FC2	0.74	(2.00)	1.01	(1.93)
		C3	-0.23	(1.06)	-0.11	(1.71)
		C4	0.45	(1.46)	0.77	(1.95)
LHC	TN congruent	F3	0.55	(2.04)	0.29	(1.88)
		F4	0.23	(1.39)	0.63	(1.59)
		FC1	0.65	(2.16)	1.00	(1.62)
		FC2	-0.47	(1.26)	0.03	(1.12)
		C3	0.52	(1.74)	0.64	(1.33)
		C4	-0.90	(1.70)	-0.37	(1.96)
	TN incongruent	F3	0.03	(2.18)	-0.51	(2.11)
		F4	-0.33	(1.86)	0.48	(2.40)
		FC1	0.73	(1.96)	0.49	(2.09)
		FC2	-0.20	(0.99)	0.23	(1.43)
		C3	0.51	(1.68)	0.18	(1.32)
		C4	-0.95	(1.53)	-1.13	(2.23)
NHC	TN congruent	F3	-0.13	(1.66)	-0.08	(1.76)
		F4	0.00	(1.32)	0.28	(1.61)
		FC1	0.19	(1.57)	0.62	(1.53)
		FC2	-0.07	(1.43)	-0.23	(1.02)
		C3	-0.15	(1.77)	0.46	(1.49)
		C4	-0.96	(1.56)	-1.21	(1.56)
	TN incongruent	F3	-0.09	(1.89)	-0.18	(1.57)
		F4	0.30	(1.66)	0.63	(1.70)
		FC1	0.75	(2.01)	0.96	(1.78)
		FC2	0.29	(1.23)	0.29	(1.55)
		C3	0.49	(1.61)	0.54	(1.57)
		C4	-0.48	(1.47)	-0.50	(2.15)

Table 6.6. Inferential statistics for the effect of UHCs, congruence, region and hemisphere on N2 peak amplitudes (μV) to TN targets. Significant effects are highlighted.

	F	***p***
Trait anger	0.32	0.57
UHC	1.09	0.34
UHC × Trait anger	0.09	0.91
Congruency	0.12	0.74
Congruency × Trait anger	0.23	0.64
Site	12.08	0.00
Site × Trait anger	0.41	0.66
Hemisphere	23.90	0.00
Hemisphere × Trait anger	1.11	0.30
UHC × Congruency	2.70	0.07
UHC × Congruency × Trait anger	1.34	0.27
UHC × Site	2.44	0.07
UHC × Site × Trait anger	0.45	0.72
Congruency × Site	2.16	0.13
Congruency × Site × Trait anger	2.26	0.12
UHC × Congruency × Site	0.78	0.53
UHC × Congruency × Site × Trait anger	1.19	0.32
UHC × Hemisphere	0.95	0.39
UHC × Hemisphere × Trait anger	2.22	0.12
Congruency × Hemisphere	1.66	0.21
Congruency × Hemisphere × Trait anger	0.97	0.33
UHC × Congruency × Hemisphere	0.83	0.44
UHC × Congruency × Hemisphere × Trait anger	1.95	0.15
Site × Hemisphere	3.23	0.06
Site × Hemisphere × Trait anger	0.21	0.77
UHC × Site × Hemisphere	1.50	0.21
UHC × Site × Hemisphere × Trait anger	0.19	0.94
Congruency × Site × Hemisphere	2.78	0.07
Congruency × Site × Hemisphere × Trait anger	0.59	0.55
UHC × Congruency × Site × Hemisphere	1.45	0.22
UHC × Congruency × Site × Hemisphere × Trait anger	0.81	0.52
	1.09	0.34

In support of prediction 7, the ANOVA revealed no main effect of UHC, anger, or congruency on N2 in response to TN word-pairs ($p > .05$). Inferential information for these results can be found in Table 6.6. However contrary to prediction 7, the ANOVA revealed a main effect of region, F (1.98, 82.98) = 12.08, $p < .05$, $np^2 = .22$, with larger N2 at frontal than frontocentral, F (1, 42) = 23.74, $p < .05$, $np^2 = .36$, or central sites, F (1, 42) = 7.95, $p < .05$, $np^2 = .16$. N2 amplitude was also larger at central than frontocentral sites, F (1, 42) = 4.21, $p < .05$, $np^2 = .09$. The ANOVA also revealed a main effect of hemisphere with significantly larger N2 in response to TN targets at left than right sites F (1.00, 42.00) = 23.90, $p < .05$, $np^2 = .36$.

Contrary to predictions 8 and 9, no N2 difference was found between high compared to low anger participants, or between RHC compared to LHC and NHC, for TN targets ($p > .05$). The ANOVA also revealed contrary to prediction 10, no N2 difference for RHC compared to LHC and NHC, for TN targets in relation to trait anger ($p > .05$). Furthermore, contrary to predictions 11 and 12, no N2 laterality difference was found between high and low anger participants, or between RHC, LHC, and NHC conditions for TN targets ($p > .05$). Inferential statistics for these results can be found in Table 6.6.

6.3.2.1.3. P3b ERP component

To explore whether compared to LHCs and NHCs, RHCs reduced P3b (reduced attentional bias towards threat) for congruent compared to incongruent TN word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (trait anger; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) x 3 (Region; centroparietal, parietal, parietooccipital) mixed ANOVA was then performed on P3b mean amplitudes to TN targets. Descriptive statistics of P3b amplitudes at each site are presented for congruent and incongruent TN targets in relation to UHC and anger group in Table 6.7. Inferential statistics of this analysis are presented in Table 6.8.

Table 6.7. Means and standard deviations of P3b mean amplitudes (μV) for TN word-pairs in relation to UHC, congruency, and anger at centroparietal, parietal and parietooccipital sites

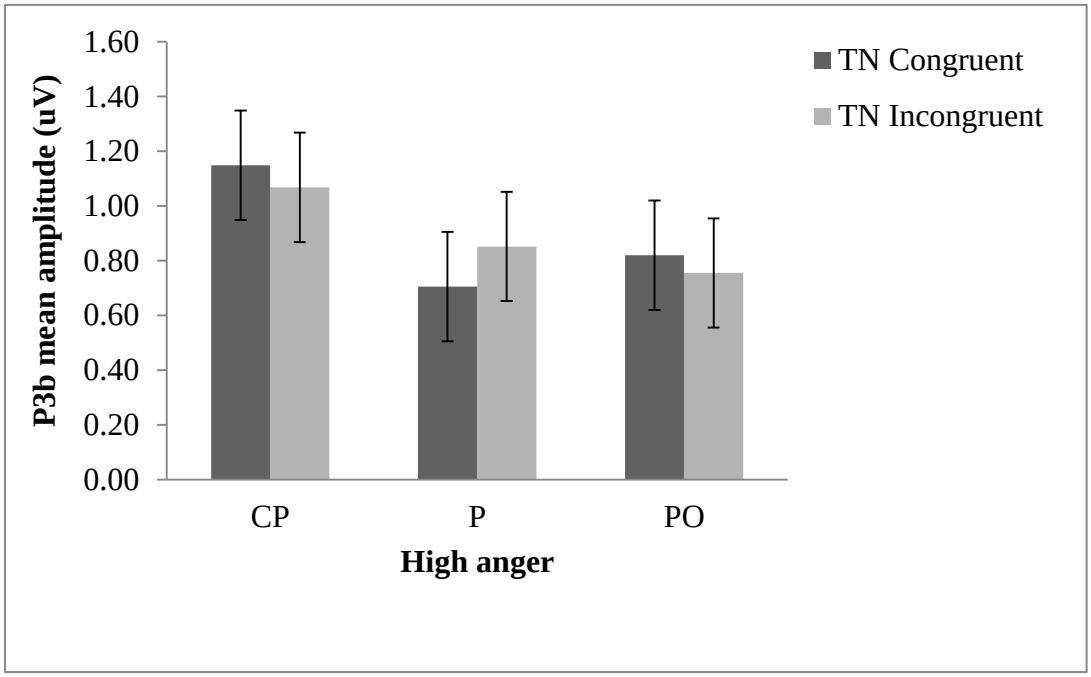
UHC	TN congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD)*	*M*	*(SD)*
RHC	TN congruent	CP1	0.98	(1.62)	1.47	(1.80)
		CP2	1.25	(1.65)	1.85	(2.03)
		P3	0.62	(1.33)	0.85	(1.71)
		P4	1.14	(1.22)	1.58	(2.03)
		PO3	0.96	(2.10)	1.42	(1.64)
		PO4	1.21	(1.71)	1.01	(1.58)
	TN incongruent	CP1	0.97	(1.38)	1.30	(1.90)
		CP2	1.26	(1.38)	1.63	(1.81)
		P3	1.15	(1.41)	0.87	(1.01)
		P4	1.26	(1.35)	1.05	(1.46)
		PO3	0.50	(1.84)	1.07	(1.23)
		PO4	0.47	(1.54)	0.46	(1.35)
LHC	TN congruent	CP1	1.18	(1.70)	1.49	(1.43)
		CP2	1.41	(1.18)	1.24	(1.73)
		P3	0.34	(1.24)	1.52	(1.45)
		P4	1.06	(1.34)	0.83	(1.60)
		PO3	0.53	(2.07)	1.24	(1.78)
		PO4	0.76	(1.81)	0.62	(2.12)
	TN incongruent	CP1	0.92	(1.22)	1.51	(1.78)
		CP2	1.43	(1.51)	1.05	(1.48)
		P3	0.76	(0.99)	0.90	(1.49)
		P4	1.20	(1.12)	0.82	(1.55)
		PO3	0.54	(1.84)	1.10	(2.02)
		PO4	1.19	(1.73)	0.85	(1.73)
NHC	TN congruent	CP1	0.77	(1.32)	1.24	(1.51)
		CP2	1.31	(1.84)	1.09	(1.61)
		P3	0.62	(1.05)	1.60	(1.49)
		P4	1.05	(1.41)	1.12	(1.59)
		PO3	0.52	(1.39)	1.25	(2.08)
		PO4	0.95	(1.68)	1.00	(2.19)
	TN incongruent	CP1	0.90	(1.41)	1.52	(1.74)
		CP2	0.93	(1.45)	1.68	(1.69)
		P3	0.34	(1.28)	1.27	(1.42)
		P4	0.41	(1.450	0.96	(1.80)
		PO3	0.37	(2.06)	1.61	(2.22)
		PO4	0.08	(1.87)	1.11	(2.65)

Table 6.8. Inferential statistics for the effect of UHC, anger, congruence, region and hemisphere on P3b mean amplitudes (μV) to TN targets. Significant effects are highlighted.

	F	***p***
Trait anger	2.96	0.95
UHC	0.35	0.70
UHC × Trait anger	1.55	0.22
Congruency	2.10	0.15
Congruency × Trait anger	0.02	0.90
Site	1.71	0.20
Site × Trait anger	0.03	0.90
Hemisphere	0.13	0.72
Hemisphere × Trait anger	2.14	0.15
UHC × Congruency	0.67	0.52
UHC × Congruency × Trait anger	3.01	0.06
UHC × Site	0.25	0.88
UHC × Site × Trait anger	1.07	0.37
Congruency × Site	1.04	0.35
Congruency × Site × Trait anger	3.45	0.04
UHC × Congruency × Site	3.42	0.02
UHC × Congruency × Site × Trait anger	0.26	0.87
UHC × Hemisphere	0.68	0.51
UHC × Hemisphere × Trait anger	2.25	0.11
Congruency × Hemisphere	0.83	0.37
Congruency × Hemisphere × Trait anger	1.39	0.25
UHC × Congruency × Hemisphere	1.22	0.30
UHC × Congruency × Hemisphere × Trait anger	0.55	0.56
Site × Hemisphere	1.85	0.17
Site × Hemisphere × Trait anger	0.56	0.54
UHC × Site × Hemisphere	1.59	0.19
UHC × Site × Hemisphere × Trait anger	0.91	0.45
Congruency × Site × Hemisphere	0.23	0.78
Congruency × Site × Hemisphere × Trait anger	0.60	0.54
UHC × Congruency × Site × Hemisphere	0.95	0.43
UHC × Congruency × Site × Hemisphere × Trait anger	0.81	0.52
	0.35	0.70

In support of prediction 7, the ANOVA revealed no main effect of UHC, anger, or congruency, site or hemisphere on P3b in response to TN targets. ($p > .05$). Inferential information for these results can be found in Table 6.8.

For prediction 8, analysis showed no significant anger × congruency interaction. However, higher order interactions including region revealed a significant anger × congruency × region interaction, F (1.73, 72.52) = 2.67, p <.05, *np2* =.08. Further analysis revealed that in low anger participants there was no P3b difference between TN congruent and incongruent targets at any site ($p > 0.05$). In high anger participants no P3b amplitude difference was observed between TN congruent and incongruent targets at centroparietal or parietooccipital sites, ($p > 0.05$) but at parietal sites P3b was significantly smaller for TN congruent than incongruent targets, t (23) -2.87, $p < 0.01$. This effect can be seen in Figures 6.5 and 6.6.



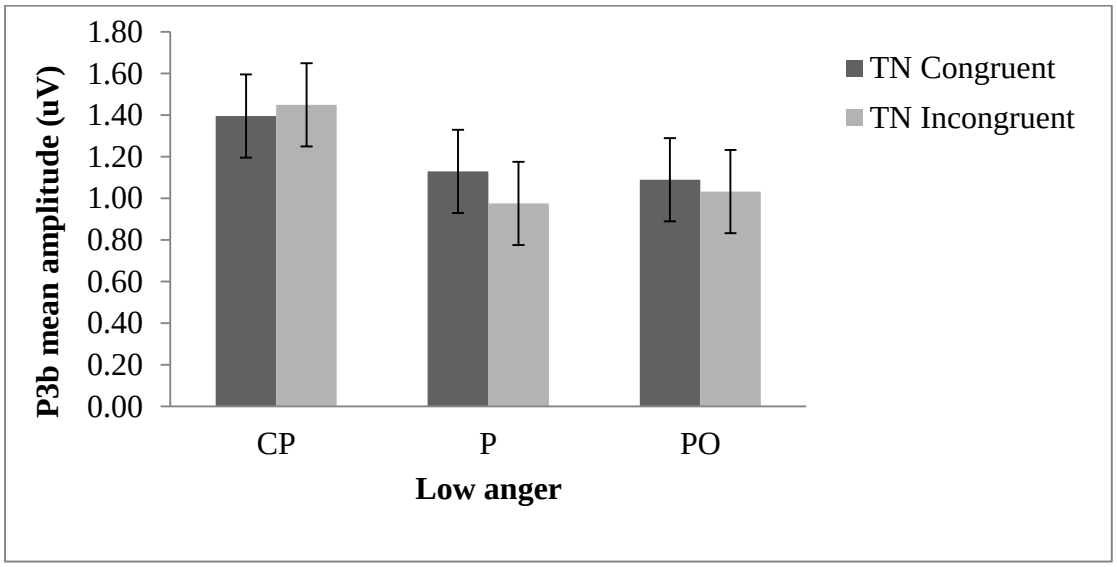


Figure 6.5. Mean parietal P3b amplitude (μV) for TN congruent compared to incongruent target in high (upper panel) and low trait anger participants (lower panel). Data are expressed as mean (SEM).

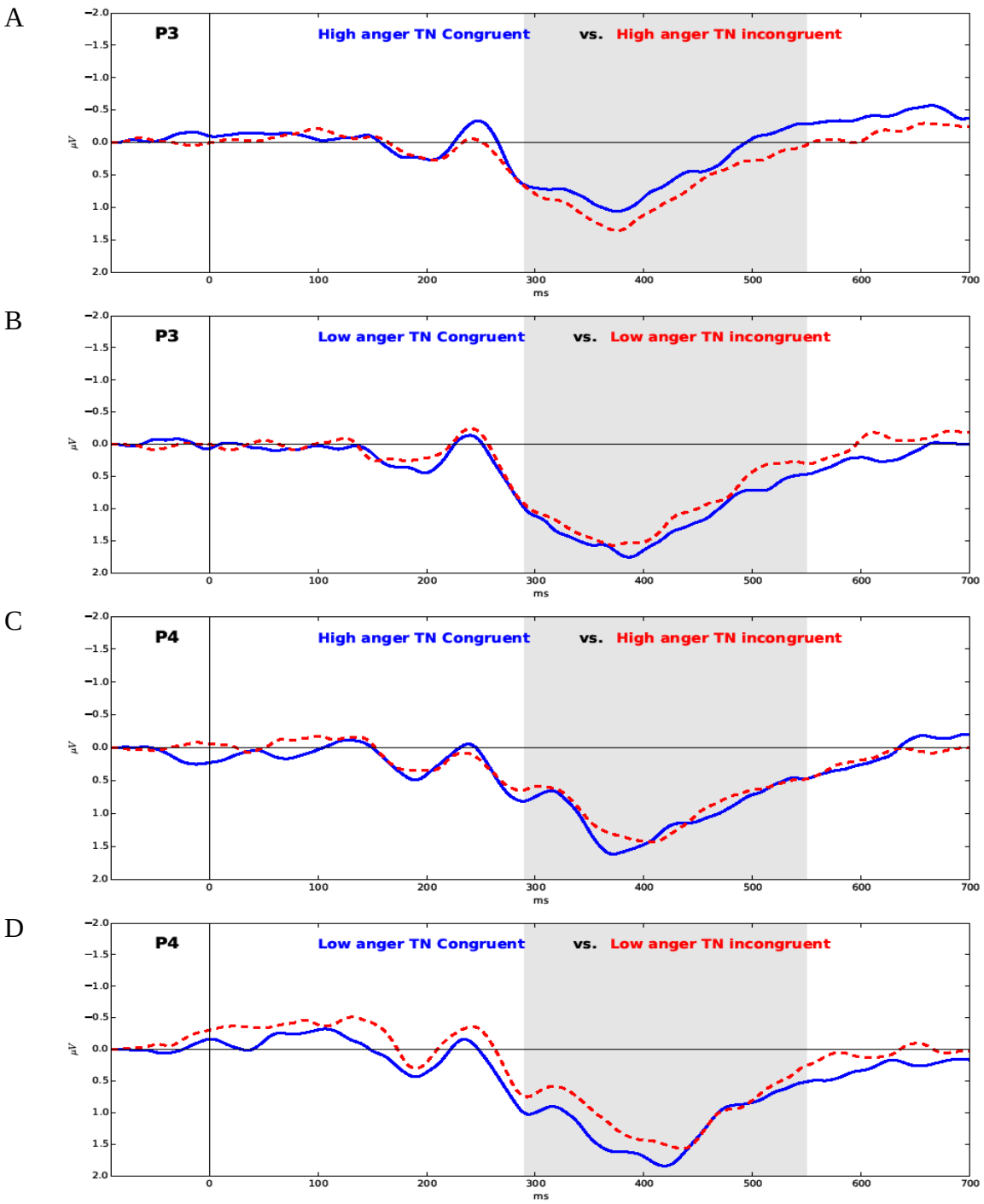
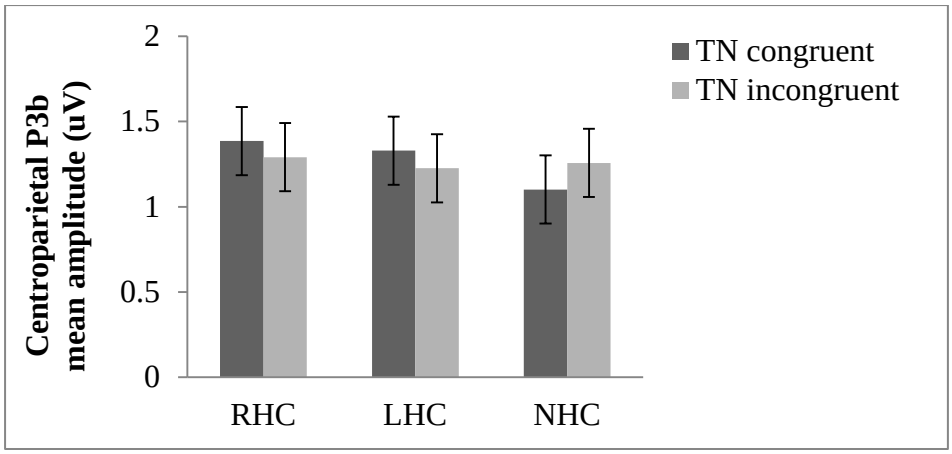


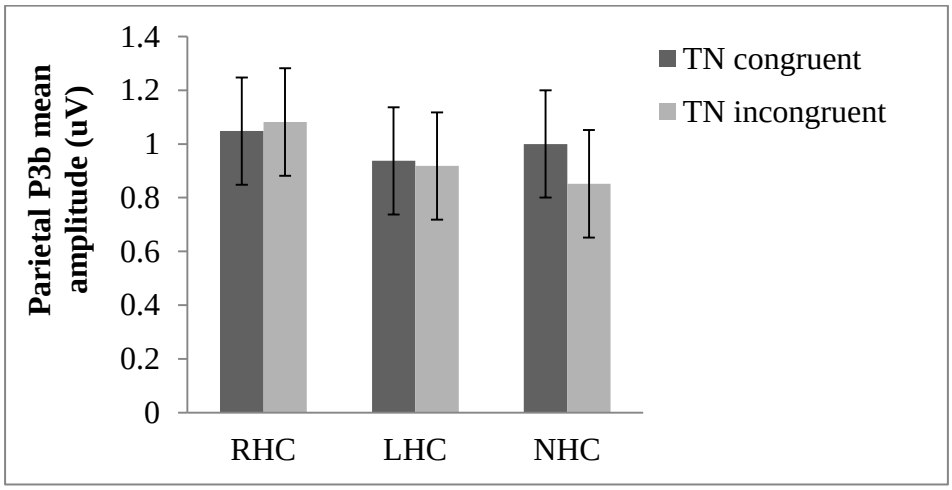
Figure 6.6. Grand average waveforms showing parietal P3b μV (shaded grey) for TN congruent compared to incongruent DPT targets in high (panels A and C) and low trait anger participants (panels B and D) at P3 and P4.

With respect to prediction 9, analysis revealed no significant UHC × congruency interaction. However, higher order interactions including region showed a significant UHC × congruency × region interaction, F (3.22, 135.18) = 3.42, p <.05, $np2$ =.08. However contrary to predictions, further analysis revealed that in the LHC condition there was no P3b difference between TN congruent and incongruent targets at centroparietal, t (43) .52, p > 0.05, parietal, t (43) -.05, p > 0.05, or at parietooccipital sites, t (43) -.81, p > 0.05 and in the NHC condition there was also no difference between TN congruent than incongruent trials at centroparietal, t (43) -.78, p > 0.05, parietal, t (43) -.12, p > 0.05, or at parietooccipital sites, t (43) -.88, p > 0.05. However, contrary to prediction 9, results showed that in the RHC condition there was no difference between TN congruent than incongruent at centroparietal, t (43) .52, p > 0.05 parietal sites, t (43) -.38, p > 0.05, but at parietooccipital sites P3b was significantly larger for TN congruent than incongruent targets, t (43) 2.81, p < 0.01. This pattern can be seen in Figures 6.7 and 6.8.

A



B



C

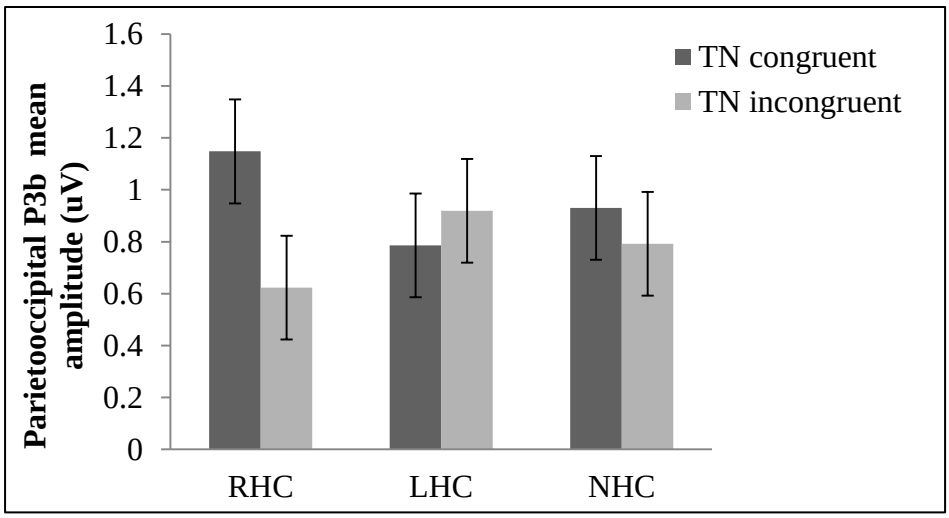


Figure 6.7. Mean parietooccipital P3b amplitude (μV) for TN congruent compared to TN incongruent DPT targets in the RHC, LHC and NHC conditions.

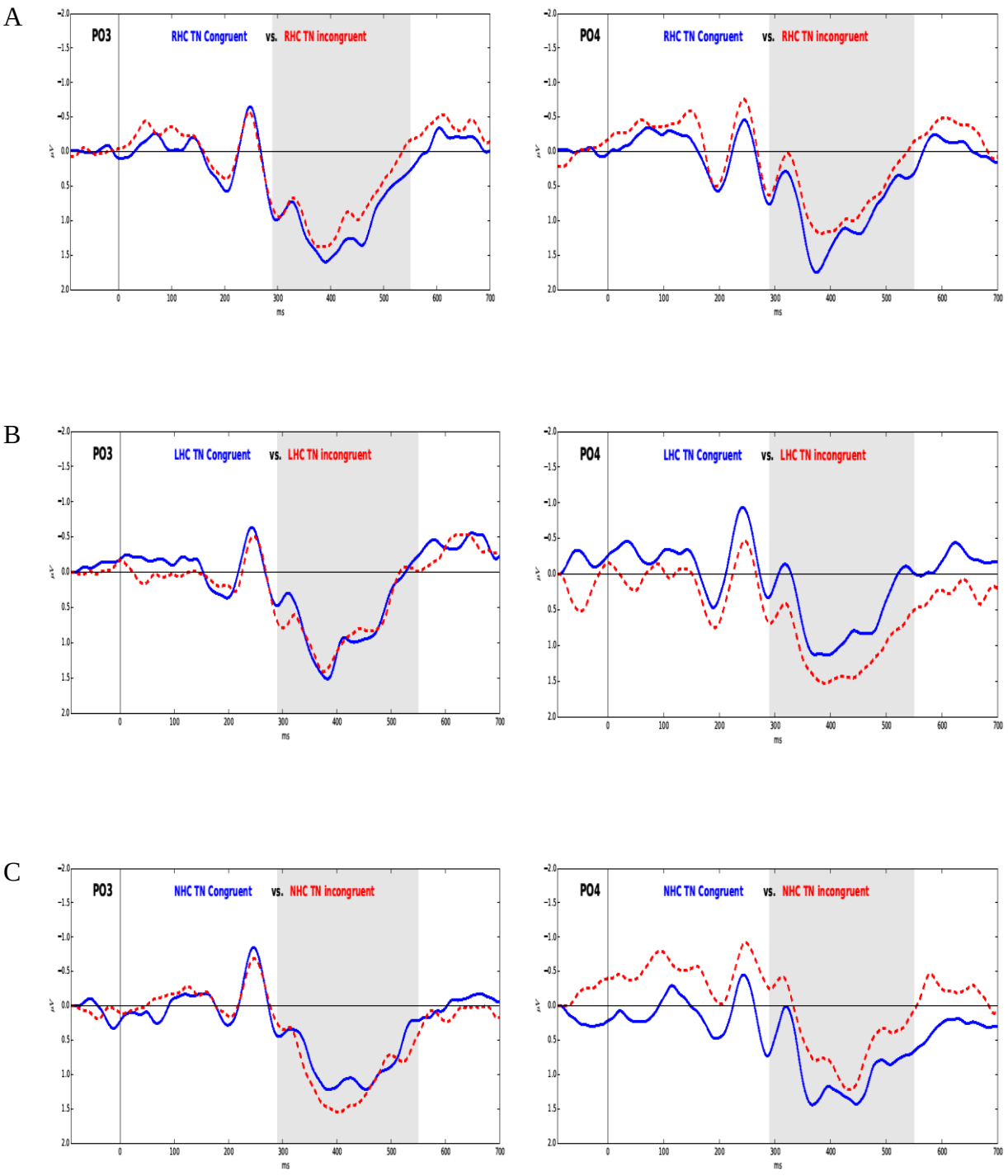


Figure 6.8. Grand average waveforms showing parietooccipital P3b μV (shaded grey) for TN congruent compared to incongruent targets in the RHC (panel A), LHC (panel B) and NHC conditions (panel C) at PO3 and PO4

The ANOVA also revealed contrary to prediction 10, no P3b difference between RHC compared to LHC and NHC, for TN targets in relation to trait anger ($p > .05$) .Contrary to predictions 11 and 12, no P3b laterality difference was found between high and low anger participants or between RHC, LHC, and NHC for TN targets ($p > .05$). Inferential statistics for these results can be found in Table 6.8.

6.3.2.2. PN word-pairs

6.3.2.2.1. P2 ERP component

To explore whether RHC compared to LHC and NHC was associated with reduced P2 (reduced attentional bias towards threat) for highly arousing threat words when presented alongside low arousal neutral words in high compared to low anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (trait anger; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) x 3 (Region; frontal, frontocentral) mixed ANOVA was performed on P2 peak amplitudes during PN trials. Means and standard deviations of P2 amplitudes at left and right frontal and frontocentral sites in relation to UHC, congruency and trait anger are presented in Table 6.9. Inferential statistics of this analysis are presented in Table 6.10.

Table 6.9. Means and standard deviations of P2 amplitudes (μV) for PN targets in relation to the UHC, congruency, and anger at left and right frontal and frontocentral sites

UHC	**PN congruence**	**Site**	**High Anger (n= 24)**		**Low anger (n=20)**	
			M	*(SD)*	*M*	*(SD*
RHC	PN congruent	F3	-0.05	(1.34)	0.01	(1.66)
		F4	0.89	(1.45)	0.74	(1.60)
		FC1	0.45	(1.61)	0.78	(1.62)
		FC2	0.91	(1.70)	1.19	(1.42)
	PN incongruent	F3	-0.07	(1.25)	0.45	(1.84)
		F4	0.24	(1.29)	1.10	(1.19)
		FC1	0.50	(1.32)	0.69	(1.72)
		FC2	0.74	(1.56)	1.06	(1.79)
LHC	PN congruent	F3	0.19	(1.22)	0.30	(1.26)
		F4	0.95	(1.18)	0.27	(2.10)
		FC1	0.50	(1.50)	0.54	(1.63)
		FC2	1.00	(1.07)	0.43	(1.81)
	PN incongruent	F3	0.33	(0.94)	0.60	(1.56)
		F4	0.72	(1.53)	0.20	(1.30)
		FC1	0.71	(1.34)	1.50	(1.74)
		FC2	0.94	(1.28)	1.08	(1.74)
NHC	PN congruent	F3	-0.05	(1.06)	0.46	(0.90)
		F4	0.59	(1.12)	0.53	(2.06)
		FC1	0.37	(1.75)	0.96	(1.36)
		FC2	0.73	(1.64)	1.09	(1.66)
	PN incongruent	F3	0.21	(1.23)	0.01	(1.64)
		F4	0.83	(1.15)	0.78	(1.05)
		FC1	1.05	(1.27)	1.06	(1.54)
		FC2	1.20	(1.27)	1.31	(1.37)

Table 6.10. Inferential statistics for the effect of UHC, trait anger, congruency, region and hemisphere on P2 peak amplitudes (μV) to PN targets. Significant effects are highlighted.

	F	***p***
Trait anger	.27	0.60
UHC	0.27	0.74
UHC × Trait anger	0.97	0.38
Congruency	2.14	0.15
Congruency × Trait anger	0.47	0.50
Site	11.31	0.00
Site × Trait anger	0.39	0.54
Hemisphere	8.33	0.01
Hemisphere × Trait anger	1.34	0.25
UHC × Congruency	0.91	0.40
UHC × Congruency × Trait anger	2.09	0.13
UHC × Site	0.86	0.42
UHC × Site × Trait anger	0.79	0.45
Congruency × Site	2.50	0.12
Congruency × Site × Trait anger	0.15	0.70
UHC × Congruency × Site	1.79	0.17
UHC × Congruency × Site × Trait anger	2.71	0.07
UHC × Hemisphere	3.01	0.06
UHC × Hemisphere × Trait anger	2.87	0.06
Congruency × Hemisphere	1.94	0.17
Congruency × Hemisphere × Trait anger	2.02	0.16
UHC × Congruency × Hemisphere	1.23	0.30
UHC × Congruency × Hemisphere × Trait anger	0.37	0.69
Site × Hemisphere	3.68	0.06
Site × Hemisphere × Trait anger	0.14	0.71
UHC × Site × Hemisphere	0.31	0.74
UHC × Site × Hemisphere × Trait anger	0.07	0.93
Congruency × Site × Hemisphere	0.05	0.83
Congruency × Site × Hemisphere × Trait anger	1.11	0.30
UHC × Congruency × Site × Hemisphere	1.23	0.30
UHC × Congruency × Site × Hemisphere × Trait anger	0.11	0.88

In support of prediction 7, the ANOVA revealed no main effect of UHC, anger, or congruency, on P2 in response to PN trials. Inferential information for these results can be found in Table 6.10. Contrary to prediction 7 however, the ANOVA revealed a significant main effect of site, with larger amplitudes at frontocentral than frontal sites, F (1.00, 42.00) =

11.31, p <.05, np^2 =.21 and a main effect of hemisphere with larger P2 amplitude at the right than the left hemisphere, F (1.00, 42.00) = 8.33, p <.05, np^2 =.17.

Further contrary to prediction 8, 9, and 10, no P2 difference was found between high and low anger participants, or between RHC compared to LHC and NHC, and no P2 amplitude difference for UHCs in relation to anger , for PN congruent than incongruent targets. Further contrary to prediction 11 and 12, no P2 laterality difference was found between high and low anger participants, or between RHC, LHC, and NHC conditions for PN targets. Inferential statistics for these results can be found in Table 6.10.

6.3.2.2.2. N2 ERP component

To explore whether compared to LHCs and NHCs, RHCs increased N2 (reduced attentional bias towards positive words) for congruent compared to incongruent TN word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (*AX-O* group; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) X 3 (Region; frontal, frontocentral, central) mixed ANOVA was then performed on N2 peak amplitudes. Means and standard deviations of N2 amplitudes at left and right frontal, frontocentral and central sites in relation to UHC, congruency and trait anger are presented in Table 6.11. *F* values and significance levels of this analysis are presented in Table 6.12.

Table 6.11. Means and standard deviations of N2 amplitudes (μV) for PN targets for UHC, congruency, and anger at left and right frontal, frontocentral and central sites

UHC	PN congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD0*	*M*	*(SD)*
RHC	PN congruent	F3	0.00	(2.31)	0.35	(2.00)
		F4	0.63	(2.30)	0.91	(2.11)
		FC1	-0.38	(1.29)	-0.18	(1.23)
		FC2	0.05	(1.34)	0.56	(1.790
		C3	-1.48	(1.82)	-0.37	(2.13)
		C4	-0.08	(1.48)	0.76	(1.52)
	PN incongruent	F3	-0.32	(1.90)	0.26	(2.16)
		F4	0.17	(1.85)	0.77	(2.01)
		FC1	-0.14	(1.02)	0.01	(1.41)
		FC2	0.37	(1.47)	0.65	(1.36)
		C3	-0.98	(1.51)	-0.47	(1.94)
		C4	0.64	(1.76)	-0.03	(1.67)
LHC	PN congruent	F3	-0.09	(1.66)	0.55	(1.99)
		F4	0.65	(1.30)	0.71	(2.32)
		FC1	-0.45	(1.29)	0.23	(1.43)
		FC2	0.29	(1.50)	0.52	(1.50)
		C3	-0.89	(1.34)	-0.48	(1.98)
		C4	0.30	(1.93)	-0.08	(1.77)
	PN incongruent	F3	0.06	(1.75)	1.27	(1.72)
		F4	0.63	(1.59)	1.15	(1.77)
		FC1	-0.32	(1.09)	0.73	(1.31)
		FC2	0.29	(1.340	0.61	(1.34)
		C3	-1.25	(1.51)	-0.92	(1.87)
		C4	0.01	(1.82)	-0.05	(2.27)
NHC	PN congruent	F3	-0.29	(2.27)	0.52	(1.57)
		F4	0.33	(2.16)	0.91	(1.94)
		FC1	-0.84	(1.56)	0.24	(1.29)
		FC2	0.31	(1.62)	0.86	(1.59)
		C3	-1.22	(1.23)	-0.91	(2.15)
		C4	0.33	(1.23)	0.47	(1.27)
	PN incongruent	F3	0.60	(1.220	0.95	(1.85)
		F4	0.75	(1.49)	1.24	(1.58)
		FC1	-0.22	(1.26)	0.47	(1.54)
		FC2	0.37	(1.36)	0.87	(1.66)
		C3	0.00	(2.31)	0.35	(2.00)
		C4	0.63	(2.30)	0.91	(2.11)

Table 6.12. Inferential statistic for the effect of UHC, congruence, region and hemisphere on N2 peak amplitudes (μV) to PN targets. Significant effects are highlighted.

	F	*p*
Trait anger	2.21	0.15
UHC	0.61	0.54
UHC × Trait anger	0.08	0.91
Congruency	2.80	0.10
Congruency × Trait anger	1.03	0.32
Site	11.18	0.00
Site × Trait anger	0.55	0.58
Hemisphere	28.81	0.00
Hemisphere × Trait anger	1.44	0.24
UHC × Congruency	1.33	0.27
UHC × Congruency × Trait anger	0.89	0.41
UHC × Site	1.65	0.18
UHC × Site × Trait anger	1.59	0.19
Congruency × Site	1.08	0.34
Congruency × Site × Trait anger	1.82	0.17
UHC × Congruency × Site	2.67	0.04
UHC × Congruency × Site × Trait anger	1.97	0.11
UHC × Hemisphere	1.44	0.24
UHC × Hemisphere × Trait anger	2.19	0.12
Congruency × Hemisphere	1.32	0.26
Congruency × Hemisphere × Trait anger	0.08	0.78
UHC × Congruency × Hemisphere	0.16	0.85
UHC × Congruency × Hemisphere × Trait anger	0.25	0.77
Site × Hemisphere	11.38	0.00
Site × Hemisphere × Trait anger	0.32	0.70
UHC × Site × Hemisphere	0.65	0.60
UHC × Site × Hemisphere × Trait anger	0.36	0.81
Congruency × Site × Hemisphere	0.81	0.44
Congruency × Site × Hemisphere × Trait anger	0.15	0.84
UHC × Congruency × Site × Hemisphere	1.45	0.23
UHC × Congruency × Site × Hemisphere × Trait anger	0.29	0.85

In support of prediction 7, the ANOVA revealed no main effect of UHC, anger, or congruency on N2 amplitudes in response to PN trials. Inferential information for these results can be found in Table 6.12. Contrary to prediction 7, the ANOVA did however reveal a significant main effect of region, F (1.98, 83.49) = 28.81, p <.05, np^2 =.21. Contrast showed that N2 was significantly larger at frontal compared to frontocentral, F (1, 42) = 22.66, p <.05, np^2 =.35 and central sites, F (1, 42) = 7.70, p <.05, np^2 =.15. No difference was found between frontocentral and central sites, F (1, 42) = 3.37, p >.05, np^2 =.09. The ANOVA also revealed a main effect of laterality with significantly greater negativity to PN targets at left than right hemisphere, F (1.00, 42.00) = 28.81, p <.05, np^2 =.41. This was qualified by a significant hemisphere × region interaction, F (1.81, 75.99) = 11.38, p <.05, np^2 =.21. Contrasts showed significantly larger negativity at left than right frontal sites but the magnitude of this difference was less at frontocentral, F (1, 42) = 23.85, <.05, np^2 =.36 and central sites, F (1, 42) = 10.29, <05, np^2 =.20. N2 amplitudes did not differ between left and right frontocentral and central sites, F (1, 42) = .70, >.05, np^2 =.02.

Contrary to predictions 8, no N2 amplitude difference was found between high and low anger participants, for PN congruent than incongruent targets (p > 0.05, see Table 6.12).

The ANOVA also revealed contrary to prediction 10, no N2 amplitude difference for RHC compared to LHC and NHC, for TN congruent than incongruent targets in relation to trait anger(p > 0.05). Further contrary to predictions 11 and 12, no N2 laterality difference was found between high and low anger participants, or between the RHC, LHC, and NHC conditions for PN trials. Inferential statistics for these results can be found in Table 6.12.

With respect to prediction 9, analysis showed no significant UHC × congruency interaction. However higher order interactions including region showed a significant UHC × congruency × region interaction, F (3.40, 142.60) = 2.67, p <.05, np^2 =.06. Contrasts revealed no N2 differences between RHC and NHC in response to congruency at frontal relative to central sites. F (1, 42) = .43, p >.05, np^2 =.01. However, a significant N2 congruency difference was observed between RHC and NHC at frontal relative to frontocentral sites, F (1, 42) = 4.31, p <.05, np^2 =.09 and at frontocentral relative to central sites, F (1, 42) = 10.28, p <.05, np^2 =.20. Contrasts also showed no N2 amplitude congruency differences between LHC and NHC at any of the sites (p >.05) and no N2 congruency differences between the RHC and LHC at frontal relative to central sites, F (1, 42) = .03, p >.05, np^2 =.00. Contrasts did however reveal a significant N2 amplitude difference between the RHC and LHC conditions for congruent compared to incongruent PN targets at frontal relative to frontocentral sites, F (1, 42) = 4.53, p <.05, np^2 =.10 and at frontocentral relative to central sites, F (1, 42) = 7.75, p <.05, np^2 =.16.

Further analysis to explore these contrasts revealed that in the RHC and LHC conditions, there was no difference between congruent and incongruent targets at any of the sites (p >0.05). In the NHC condition, no difference was found between congruent and incongruent trials at frontal or at central sites, (p >0.05) but at frontocentral sites N2 was significantly smaller for congruent than incongruent PN targets, t (43) -2.09, p < 0.05. However, following Bonferroni corrections this difference failed to reach significance.

Furthermore, no N2 amplitude difference between RHC and LHC for congruent or incongruent PN targets at frontal or central sites was observed, (p >0.05). There was also no N2 amplitude difference between the RHC and LHC condition for congruent PN targets at

frontocentral sites, t (43) .07, p >0.05 but for incongruent PN targets, frontocentral N2 was significantly smaller in the RHC than LHC condition, t (43) -2.15, p < 0.05. However following bonferroni adjustments this difference again failed to reach significance.

Further analysis also showed no N2 amplitude difference between the RHC and NHC conditions for congruent or incongruent PN targets at frontal or central sites, (p >0.05). At frontocentral sites no N2 UHC amplitude difference was observed for congruent targets, t (43) .64, p >0.05, but for incongruent PN targets frontocentral N2 was significantly smaller in the RHC than the NHC condition, t (43) -3.07, p < 0.01. This pattern can be seen in Figures 6.9 and 6.10.

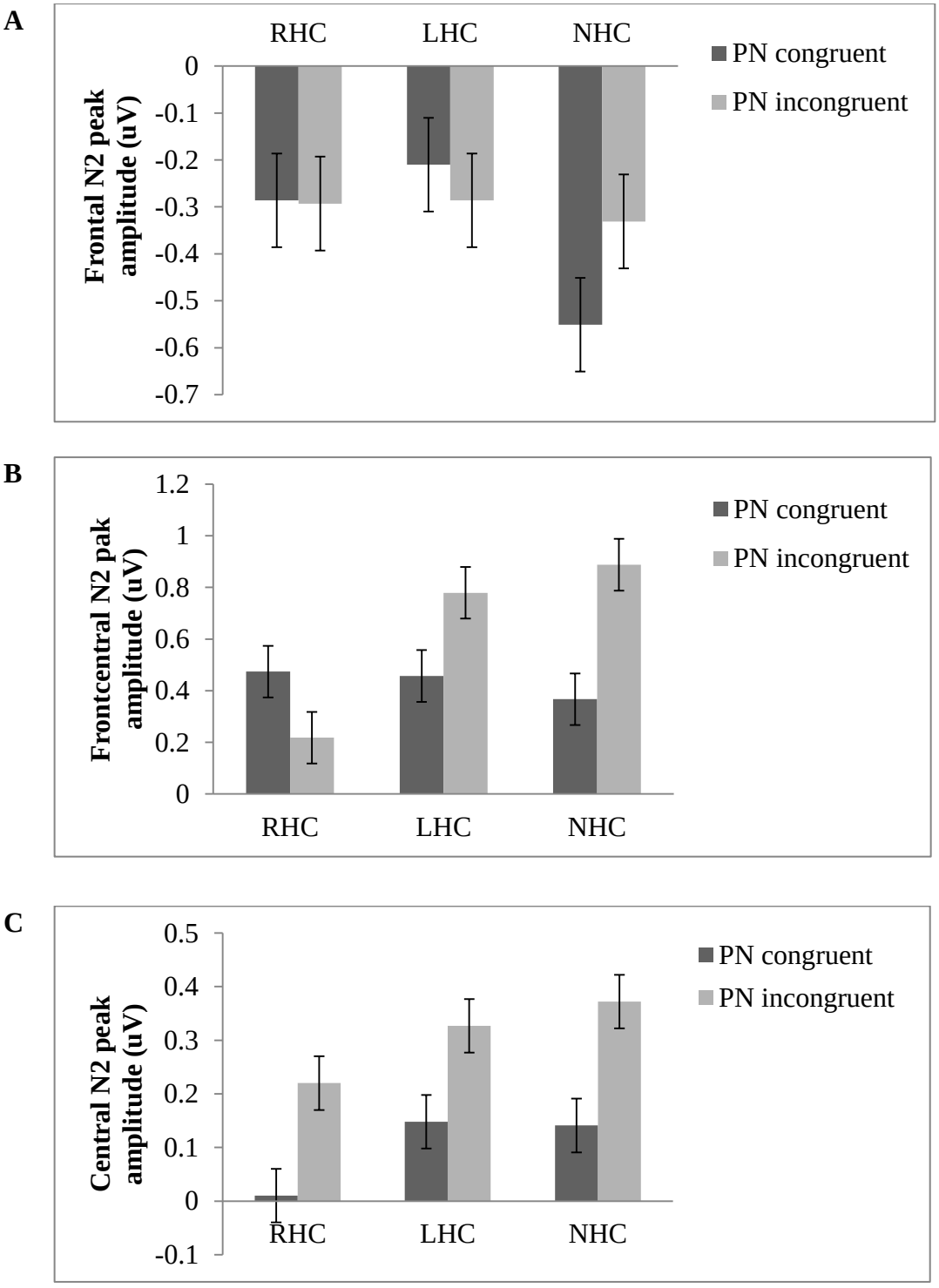


Figure 6.9. Mean N2 amplitude (μV) for PN congruent and incongruent DPT targets in the RHC compared to LHC and NHC conditions at frontal (panel A), frontocentral (panel B) and central sites (panel C). Data are expressed as mean (SEM)

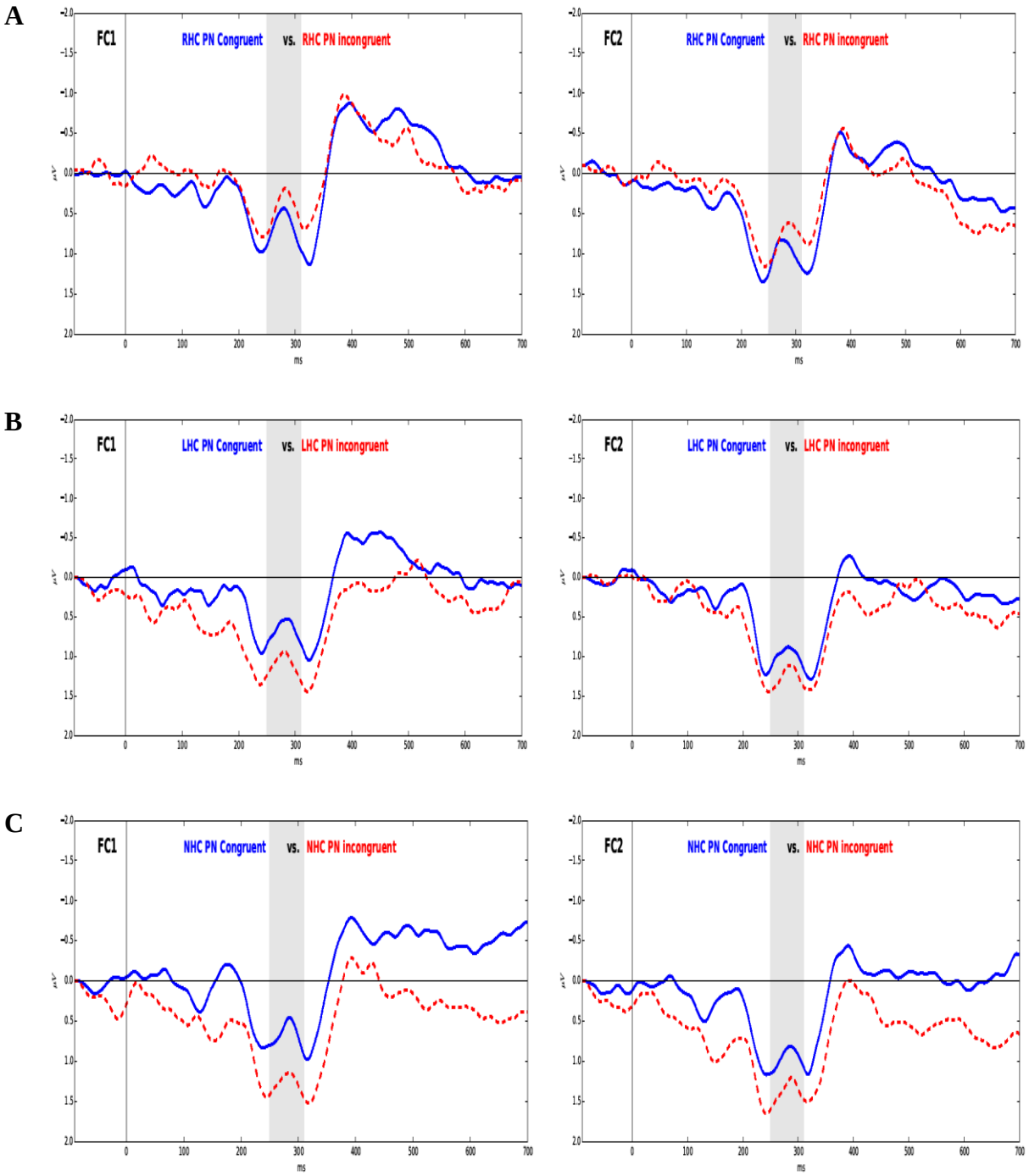


Figure 6.10. Grand average waveforms showing frontocentral N2 μV (shaded grey) for RHC (panel A), LHC (panel B) and NHC conditions (panel C) for PN congruent (solid line) and incongruent DPT targets (dashed line) at FC1 and FC2 (left and right waveforms respectively). Illustrates smaller N2 for PN incongruent in the RHC compared to the NHC and LHC conditions

6.3.2.2.3. P3b ERP component

To explore whether compared to LHCs and NHCs, RHCs reduced P3b (reduced attentional bias towards threat) for congruent compared to incongruent PN word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (Trait anger; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) x 3 (Region; centroparietal, parietal, parietooccipital) mixed ANOVA was performed on P3b mean amplitudes during PN trials. Means and standard deviations of P3b amplitudes are presented for congruent and incongruent PN targets in relation to UHC for high and low trait anger participants in Table 6.13. *F* values and significance levels of this analysis are presented in Table 6.14.

Table 6.13. Means and standard deviations of P3b amplitudes (μV) for PN targets in relation to the UHC, congruency, and trait anger at left and right centroparietal, parietal and parietooccipital sites

UHC	PN congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD)*	*M*	*(SD)*
RHC	PN congruent	CP1	0.90	(1.43)	1.42	(1.67)
		CP2	1.20	(1.41)	1.68	(1.86)
		P3	0.91	(1.22)	1.36	(1.29)
		P4	1.12	(1.01)	1.45	(1.63)
		PO3	0.86	(1.73)	1.21	(2.34)
		PO4	1.32	(1.50)	0.87	(1.39)
	PN incongruent	CP1	1.32	(1.32)	1.53	(1.33)
		CP2	1.51	(1.06)	1.59	(1.71)
		P3	1.18	(1.17)	1.06	(1.44)
		P4	1.46	(1.11)	0.98	(1.50)
		PO3	0.57	(1.76)	0.98	(1.77)
		PO4	0.90	(1.53)	0.96	(1.38)
LHC	PN congruent	CP1	1.18	(1.25)	1.23	(1.66)
		CP2	1.22	(1.13)	1.16	(1.88)
		P3	0.69	(0.98)	0.75	(1.40)
		P4	0.81	(1.33)	0.95	(1.18)
		PO3	0.27	(1.88)	0.58	(1.82)
		PO4	0.67	(2.02)	0.74	(1.97)
	PN incongruent	CP1	1.38	(1.60)	1.76	(1.53)
		CP2	1.32	(1.51)	1.23	(1.41)
		P3	0.66	(1.56)	1.19	(1.24)
		P4	1.06	(1.01)	0.95	(1.62)
		PO3	0.04	(2.44)	1.08	(1.48)
		PO4	0.91	(1.78)	0.67	(2.16)
NHC	PN congruent	CP1	1.03	(1.96)	1.55	(1.42)
		CP2	1.37	(1.54)	1.14	(1.35)
		P3	0.27	(1.36)	0.75	(1.76)
		P4	0.86	(1.92)	1.01	(1.58)
		PO3	0.50	(2.20)	0.63	(2.12)
		PO4	0.36	(2.13)	0.81	(2.18)
	PN incongruent	CP1	1.35	(1.68)	1.68	(1.69)
		CP2	1.21	(1.19)	1.35	(1.67)
		P3	0.58	(1.07)	0.71	(1.61)
		P4	0.79	(1.42)	0.95	(1.08)
		PO3	0.34	(1.54)	0.90	(1.97)
		PO4	0.52	(1.62)	0.40	(1.32)

Table 6.14. Inferential statistics for the effect of UHC, congruence, region and hemisphere on P3b mean amplitudes (μV) to PN targets. Significant effects are highlighted.

	F	***p***
Trait anger	1.00	1.00
UHC	2.85	0.07
UHC × Trait anger	0.02	0.97
Congruency	1.13	0.29
Congruency × Trait anger	0.22	0.64
Site	4.32	0.04
Site × Trait anger	0.02	0.91
Hemisphere	0.45	0.51
Hemisphere × Trait anger	1.52	0.22
UHC × Congruency	0.50	0.59
UHC × Congruency × Trait anger	0.60	0.54
UHC × Site	0.85	0.47
UHC × Site × Trait anger	0.30	0.84
Congruency × Site	1.49	0.23
Congruency × Site × Trait anger	1.43	0.25
UHC × Congruency × Site	0.27	0.84
UHC × Congruency × Site × Trait anger	0.77	0.51
UHC × Hemisphere	0.07	0.91
UHC × Hemisphere × Trait anger	0.06	0.92
Congruency × Hemisphere	0.69	0.41
Congruency × Hemisphere × Trait anger	1.39	0.25
UHC × Congruency × Hemisphere	0.08	0.91
UHC × Congruency × Hemisphere × Trait anger	1.06	0.35
Site × Hemisphere	1.18	0.30
Site × Hemisphere × Trait anger	0.72	0.46
UHC × Site × Hemisphere	1.69	0.17
UHC × Site × Hemisphere × Trait anger	0.60	0.62
Congruency × Site × Hemisphere	0.57	0.56
Congruency × Site × Hemisphere × Trait anger	1.58	0.21
UHC × Congruency × Site × Hemisphere	0.09	0.97
UHC × Congruency × Site × Hemisphere × Trait anger	1.99	0.12

In support of prediction 7, the ANOVA revealed no main effect of UHCs, anger, congruency or hemisphere on P3b for PN word-pairs. Inferential information for these results can be found in Table 6.14. However contrary to prediction 7, the ANOVA did reveal a significant main effect of region, F (1.16, 47.40) = 4.32, p <.05, np^2 =.10. Contrast showed that P3b amplitude was significantly larger at centroparietal compared to central, F (1, 42) = 5.24, p <.05, np^2 =.11 and parietooccipital sites, F (1, 42) = 4.57, p <.05, np^2 =.10. No difference was found between centroparietal and central sites, F (1, 42) = 2.27, p >05, np^2 =.05.

Contrary to predictions 8 and 9, no P3b difference was found between high and low anger participants, or between RHCs compared to LHCs and NHCs, for PN congruent compared to incongruent targets. The ANOVA also revealed contrary to prediction 10, no P3b difference for RHCs compared to LHCs and NHCs in relation to trait anger for PN congruent compared incongruent targets. Furthermore, contrary to predictions 11 and 12, no P3b laterality difference was found between high and low anger participants or between RHCs, LHCs, and NHCs for PN trials. Inferential statistics for these results can be found in Table 6.1.4.

6.3.2.3. TP word-pairs

6.3.2.3.1. P2 ERP component

To explore whether compared to LHCs and NHCs, RHCs increased P2 (reduced attentional bias towards threat) for congruent compared to incongruent TP word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (Trait anger; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) x 3 (Region; frontal, frontocentral) mixed ANOVA was performed on P2 peak amplitudes during TP trials. Means and standard deviations of P2 amplitudes at left and right frontal and frontocentral sites in relation to for TP congruency, UHC condition and trait anger, are presented in Table 6.15. *F* values and significance levels of this analysis are presented in Table 6.16.

Table 6. 15. Means and standard deviations of P2 amplitudes (μV) for TP targets in relation to the UHC, congruency, and anger at left and right frontal and frontocentral sites

UHC	TP congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD)*	*M*	*(SD)*
RHC	TP congruent	F3	0.40	(1.32)	0.70	(1.24)
		F4	0.70	(1.36)	0.40	(1.93)
		FC1	0.88	(1.53)	1.13	(1.42)
		FC2	1.18	(1.32)	1.06	(1.55)
	TP incongruent	F3	0.32	(1.17)	0.38	(1.69)
		F4	0.38	(1.42)	0.67	(1.53)
		FC1	0.53	(1.32)	0.87	(1.68)
		FC2	0.98	(1.07)	0.89	(1.75)
LHC	TP congruent	F3	-0.02	(1.07)	0.11	(1.06)
		F4	0.65	(1.82)	0.82	(1.30)
		FC1	0.66	(1.17)	0.98	(1.59)
		FC2	1.13	(1.48)	1.47	(1.72)
	TP incongruent	F3	0.01	(1.15)	0.26	(1.28)
		F4	0.28	(1.41)	0.31	(1.66)
		FC1	0.54	(1.31)	0.64	(1.64)
		FC2	1.22	(1.23)	0.75	(1.58)
NHC	TP congruent	F3	0.17	(1.10)	0.82	(1.62)
		F4	0.41	(1.65)	0.74	(1.43)
		FC1	0.40	(1.26)	1.18	(1.40)
		FC2	0.57	(1.38)	1.31	(1.12)
	TP incongruent	F3	0.56	(1.05)	0.28	(1.37)
		F4	0.46	(1.22)	0.49	(1.26)
		FC1	0.85	(1.28)	0.53	(1.37)
		FC2	0.94	(1.29)	1.05	(1.13)

Table 6.16. Inferential statistics for the effect of UHC, congruence, region and hemisphere on P2 peak amplitudes (μV) to TP targets. Significant effects are highlighted.

	F	***p***
Trait anger	0.40	0.53
UHC	0.35	0.70
UHC × Trait anger	0.13	0.88
Congruency	4.35	0.04
Congruency × Trait anger	4.07	0.05
Site	22.35	0.00
Site × Trait anger	0.02	0.88
Hemisphere	3.87	0.06
Hemisphere × Trait anger	0.27	0.61
UHC × Congruency	0.10	0.90
UHC × Congruency × Trait anger	2.27	0.11
UHC × Site	1.70	0.19
UHC × Site × Trait anger	0.45	0.63
Congruency × Site	0.10	0.75
Congruency × Site × Trait anger	1.07	0.31
UHC × Congruency × Site	0.51	0.57
UHC × Congruency × Site × Trait anger	0.54	0.56
UHC × Hemisphere	2.21	0.12
UHC × Hemisphere × Trait anger	0.57	0.55
Congruency × Hemisphere	0.00	0.96
Congruency × Hemisphere × Trait anger	0.53	0.47
UHC × Congruency × Hemisphere	1.46	0.24
UHC × Congruency × Hemisphere × Trait anger	1.67	0.20
Site × Hemisphere	0.34	0.56
Site × Hemisphere × Trait anger	0.13	0.72
UHC × Site × Hemisphere	0.19	0.81
UHC × Site × Hemisphere × Trait anger	0.36	0.69
Congruency × Site × Hemisphere	1.23	0.28
Congruency × Site × Hemisphere × Trait anger	1.93	0.17
UHC × Congruency × Site × Hemisphere	0.77	0.46
UHC × Congruency × Site × Hemisphere × Trait anger	0.64	0.52

In support of prediction 7, the ANOVA revealed no main effect of UHC, anger, or hemisphere on P2 for TP word-pairs. Inferential information for these results can be found in Table 6.16. However, a significant main effect of site emerged, with larger P2 at frontocentral than frontal sites, F (1.00, 41.00) = 22.35, p <.05, np^2 =.35. A significant main effect of congruency also emerged with larger P2 for TP congruent than incongruent targets, F (1.00, 41.00) = 4.35, p <.05, np^2 =.10.

With regards to prediction 8 that in high compared to low anger participants, P2 would be larger threat congruent than incongruent trials the ANOVA revealed a trait anger × congruency trend for TP word-pairs, F (1.00, 41.00) = 4.07, p = 0.05, np^2 = .09. However contrary to predictions, additional analysis revealed that in high anger participants there was no significant P2 amplitude difference between TP congruent and incongruent targets, t (23) -.00, p > 0,05. Interestingly, in low anger participants P2 was significantly larger for TP congruent than incongruent trials, t (19) 2.16, p < 0.05. However, following Bonferroni adjustments this difference failed to reach significance.

Contrary to predictions 9 and 10, the ANOVA showed no P2 differences for RHC compared to LHC and NHC, for TP congruent than incongruent targets in isolation or relation to trait anger. Contrary to predictions 11 and 12, no P2 laterality difference was found between high and low anger participants, or between RHCs, compared to LHCs and NHCs in TP trials. Inferential statistics for these results can be found in Table 6.16.

6.3.2.3.2. N2 ERP component

To explore whether compared to LHCs and NHCs, RHCs increased N2 (reduced attentional bias towards threat) for congruent compared to incongruent TP word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (*AX-O* group; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) X 3 (Region; frontal, frontocentral, central) mixed ANOVA was performed on N2 peak amplitudes during TP trials. Means and standard deviations of N2 amplitudes at left and right frontal, frontocentral and central sites in relation to for TP congruency, UHC condition and trait anger group, are presented in Table 6. 17. *F* values and significance levels of this analysis are presented in Table 6.18.

Table 6.17. Means and standard deviations of N2 amplitudes (μV) for TP targets for UHCs, congruency, and trait anger at left and right frontal, frontocentral and central sites

UHC	TP congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD)*	*M*	*(SD)*
RHC	TP congruent	F3	-0.78	(1.77)	-0.77	(1.83)
		F4	0.43	(1.75)	-0.06	(2.04)
		FC1	0.36	(1.69)	0.51	(1.70)
		FC2	0.87	(1.82)	0.79	(1.86)
		C3	-0.59	(1.27)	0.08	(1.37)
		C4	0.64	(1.49)	0.75	(1.70)
	TP incongruent	F3	-0.78	(1.34)	-0.86	(1.85)
		F4	-0.15	(1.76)	0.20	(2.18)
		FC1	-0.06	(1.72)	0.37	(2.09)
		FC2	0.53	(1.92)	0.65	(2.04)
		C3	-0.50	(1.02)	0.21	(1.56)
		C4	0.26	(1.35)	0.52	(1.33)
LHC	TP congruent	F3	-1.09	(1.43)	-0.90	(1.89)
		F4	0.37	(2.18)	0.58	(1.78)
		FC1	0.12	(1.38)	0.63	(1.84)
		FC2	1.04	(2.01)	1.37	(1.91)
		C3	-0.23	(1.22)	-0.26	(1.31)
		C4	0.61	(1.82)	0.39	(1.54)
	TP incongruent	F3	-1.01	(1.75)	-0.88	(1.87)
		F4	-0.42	(2.09)	0.25	(1.66)
		FC1	-0.37	(1.89)	0.82	(1.52)
		FC2	0.41	(2.14)	1.00	(1.45)
		C3	-0.64	(1.11)	0.50	(1.57)
		C4	0.18	(1.60)	1.13	(1.49)
NHC	TP congruent	F3	-0.84	(1.29)	-0.72	(2.38)
		F4	-0.38	(1.82)	-0.40	(2.160
		FC1	-0.24	(1.38)	0.31	(1.55)
		FC2	0.16	(1.76)	0.31	(1.94)
		C3	-0.21	(1.19)	0.17	(1.73)
		C4	0.12	(1.62)	0.57	(1.75)
	TP incongruent	F3	-0.69	(1.27)	-1.12	(1.90)
		F4	0.02	(1.47)	0.37	(1.30)
		FC1	0.39	(1.15)	-0.08	(1.72)
		FC2	0.59	(1.70)	0.71	(1.63)
		C3	-0.35	(1.32)	0.03	(1.41)
		C4	0.39	(.53)	0.86	(1.42)

Table 6.18. Inferential statistics for the effect of UHC, trait anger, congruence, region and hemisphere on N2 peak amplitudes (μV) to TP targets. Significant effects are highlighted.

	F	***p***
Trait anger	2.21	0.15
UHC	0.85	0.43
UHC × Trait anger	1.03	0.36
Congruency	0.12	0.73
Congruency × Trait anger	1.27	0.27
Site	16.22	0.00
Site × Trait anger	0.67	0.50
Hemisphere	25.19	0.00
Hemisphere × Trait anger	0.06	0.81
UHC × Congruency	1.46	0.24
UHC × Congruency × Trait anger	1.66	0.20
UHC × Site	1.20	0.32
UHC × Site × Trait anger	0.71	0.58
Congruency × Site	0.63	0.52
Congruency × Site × Trait anger	0.95	0.39
UHC × Congruency × Site	1.65	0.18
UHC × Congruency × Site × Trait anger	1.41	0.24
UHC × Hemisphere	1.05	0.35
UHC × Hemisphere × Trait anger	0.58	0.56
Congruency × Hemisphere	0.00	0.98
Congruency × Hemisphere × Trait anger	0.96	0.33
UHC × Congruency × Hemisphere	4.26	0.02
UHC × Congruency × Hemisphere × Trait anger	0.55	0.58
Site × Hemisphere	3.26	0.05
Site × Hemisphere × Trait anger	0.74	0.47
UHC × Site × Hemisphere	0.37	0.81
UHC × Site × Hemisphere × Trait anger	0.28	0.87
Congruency × Site × Hemisphere	0.00	1.00
Congruency × Site × Hemisphere × Trait anger	1.77	0.18
UHC × Congruency × Site × Hemisphere	1.68	0.16
UHC × Congruency × Site × Hemisphere × Trait anger	0.71	0.57

In support of prediction 7, the ANOVA no main effect of UHCs, anger, or congruency on N2 in response to TP word-pairs. Inferential information for these results can be found in Table 6.18. However, the ANOVA did reveal a significant main effect of region, F (1.75, 73.44) = 16.22, p <.05, np^2 =.28. Contrast revealed that N2 was significantly larger at frontal compared to frontocentral, F (1, 42) = 40.58, p <.05, np^2 =.49 and central sites, F (1, 42) = 10.52, p <.05, np^2 =.20. No difference was found between frontocentral and central sites, F

(1, 42) = 3.64, p >.05, np^2 =.08. The ANOVA also revealed a significant, main effect of hemisphere with significantly larger N2 for TP targets at the left than the right hemisphere, F (1.00, 42.00) = 25.19, p <.05, np^2 =.38. This was qualified by a region × hemisphere trend, F (1.75, 73.73) = 3.26, p =.05, np^2 =.07. Contrasts revealed no laterality difference between frontal and central sites, F (1, 42) = 1.41, p >.05 or between frontocentral and central sites, F (1, 42) = 1.71, p >.05. However, a significant laterality difference was revealed between frontal and frontocentral sites, F (1, 42) = 7.67, p < .01. At frontocentral sites there was no laterality difference but at frontal sites N2 was larger at the left than the right hemisphere.

Contrary to predictions 8, no significant N2 amplitude difference was found between high and low anger participants, for TP congruent than incongruent targets (p > 0.05, see Table 6.18).

With respect to prediction 9, analysis revealed no significant N2 amplitude UHC × congruency interaction. However, higher order interactions including hemisphere revealed a significant UHC × congruency × hemisphere interaction, F (198, 82.97) = 2.67, p <.05, np^2 =.09. Contrasts showed a significant N2 laterality difference between the RHC and NHC conditions for TP congruent compared to incongruent targets, F (1, 42) = 4.41, p <05, np^2 =.10. In the RHC condition N2 was larger for TP incongruent than congruent targets in the right hemisphere but in the left hemisphere N2 did not differ in response to congruency. In the NHC condition, N2 was larger for congruent than incongruent targets in the right hemisphere but again in the left hemisphere there was no congruency effect. Contrasts also showed a significant N2 difference between the LHC and NHC conditions for TP congruent and incongruent responses in the left and right hemispheres , F (1, 42) = 7.63, p <05, np^2 =.15. Similar to the RHC condition, in the LHC condition N2 was larger for incongruent than

congruent targets in the right hemisphere but in the left hemisphere there was no effect of congruency. The similarity between laterality patterns in the RHC compared to the LHC condition was further illustrated in a contrast that revealed no N2 laterality difference n in the RHC and LHC conditions for TP congruent compared to incongruent targets, F (1, 42) = 3.46, p >.05, np^2 =.01. This pattern can be seen in Figures 6.11, 6.12, 6.13. and 6.14.

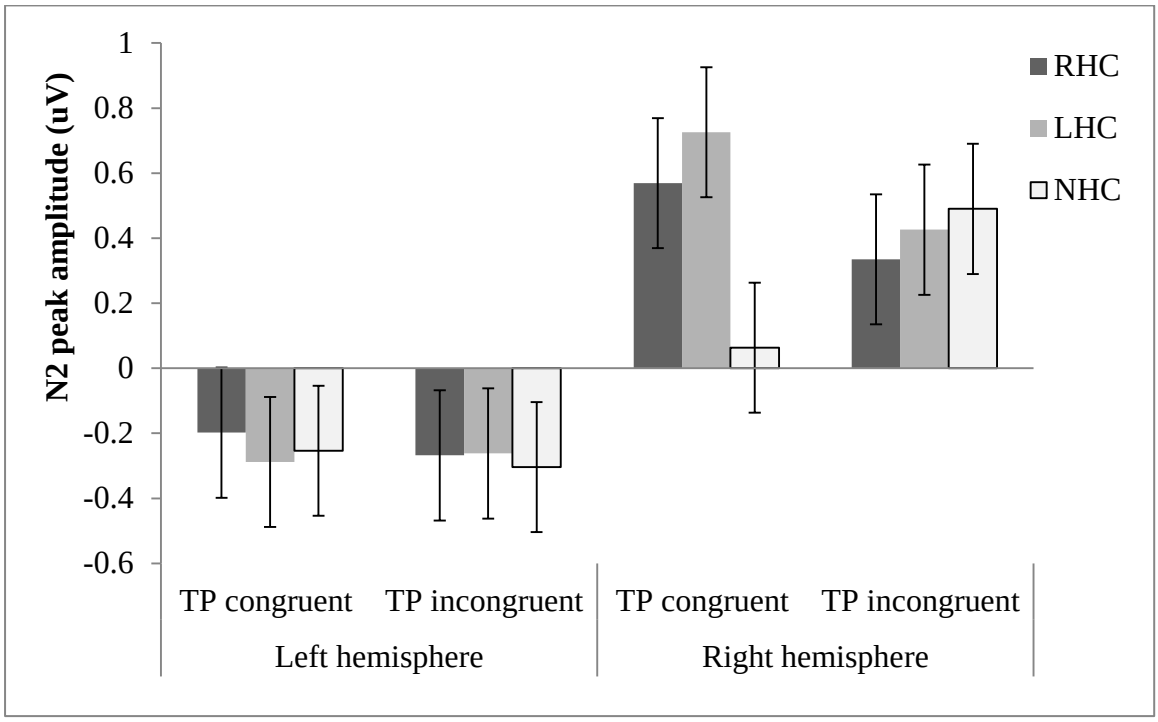


Figure 6.11. Mean N2 peak amplitude (μV) for TP congruent and incongruent DPT targets at left and right frontal, frontocentral and central sites. Data are expressed as mean (SEM)

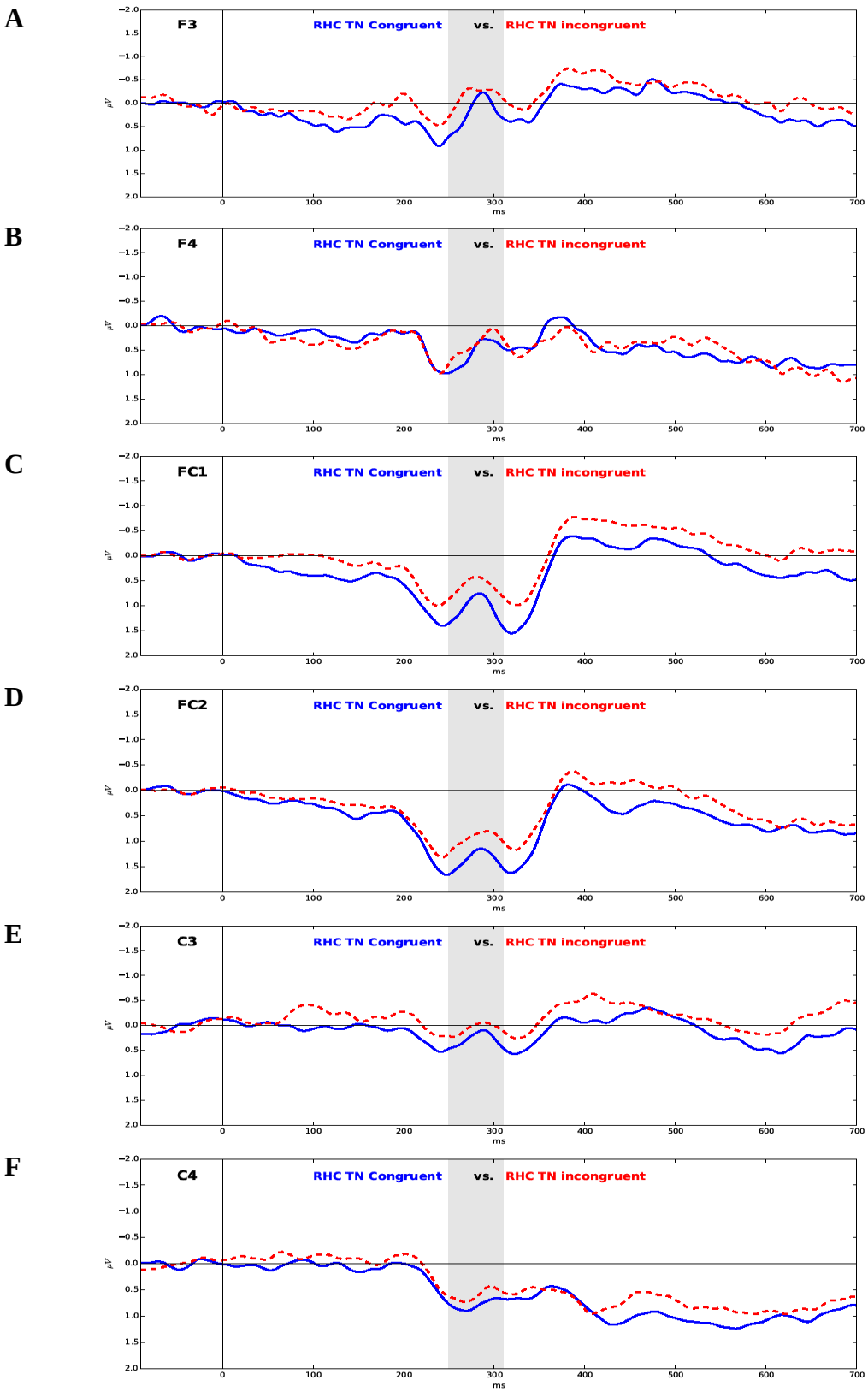


Figure 6.12. Grand average waveforms showing N2 µV (shaded grey) for TP congruent vs. incongruent DPT targets in the RHC condition at left compared to right frontal (panel A and B), frontocentral (panel C and D) and central sites (panels E and F)

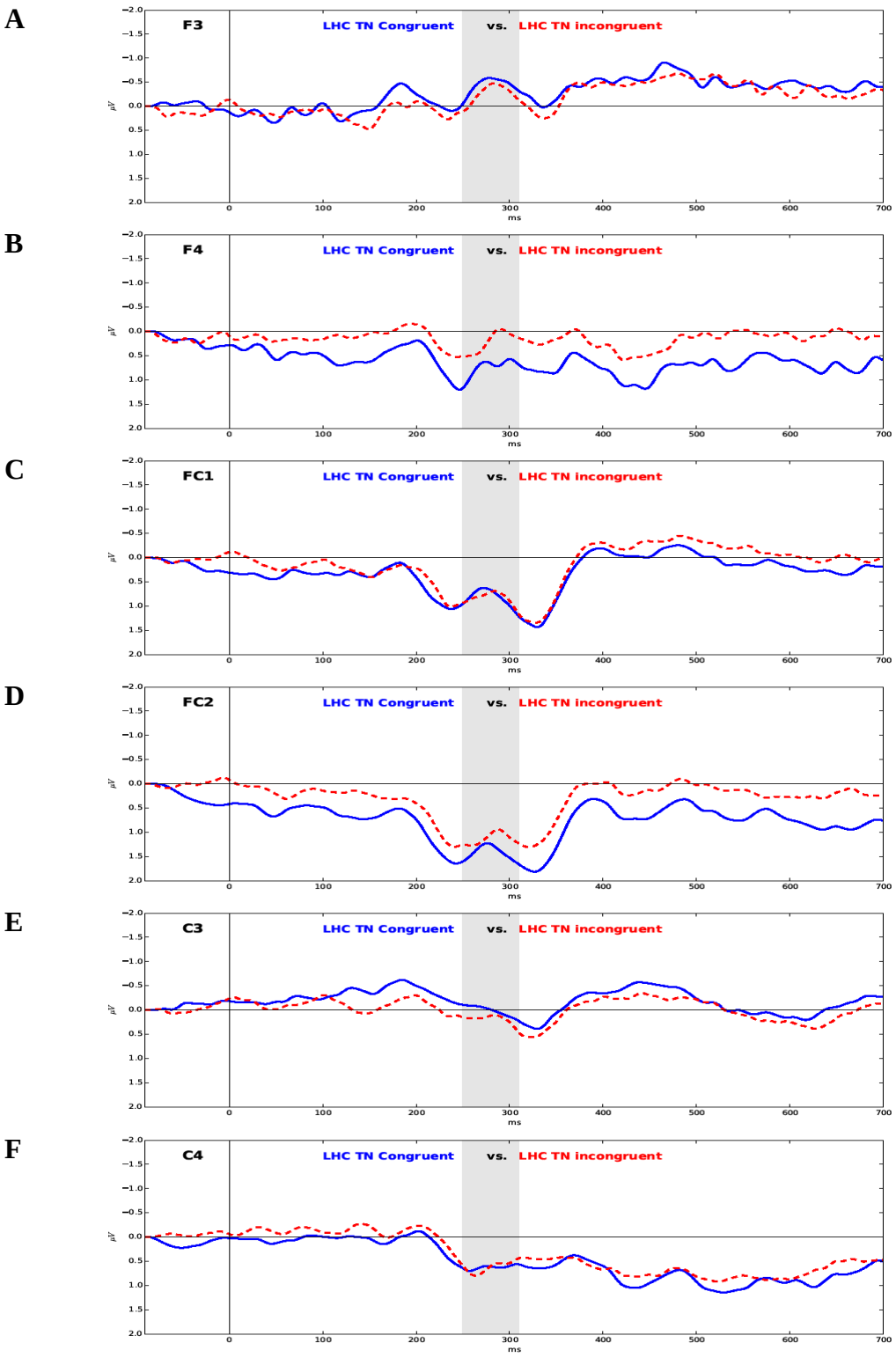


Figure 6.13. Grand average waveforms showing N2 μV (shaded grey) for TP congruent vs. incongruent DPT targets in the LHC condition at left compared to right frontal (panel A and B), frontocentral (panel C and D) and central sites (panels E and F)

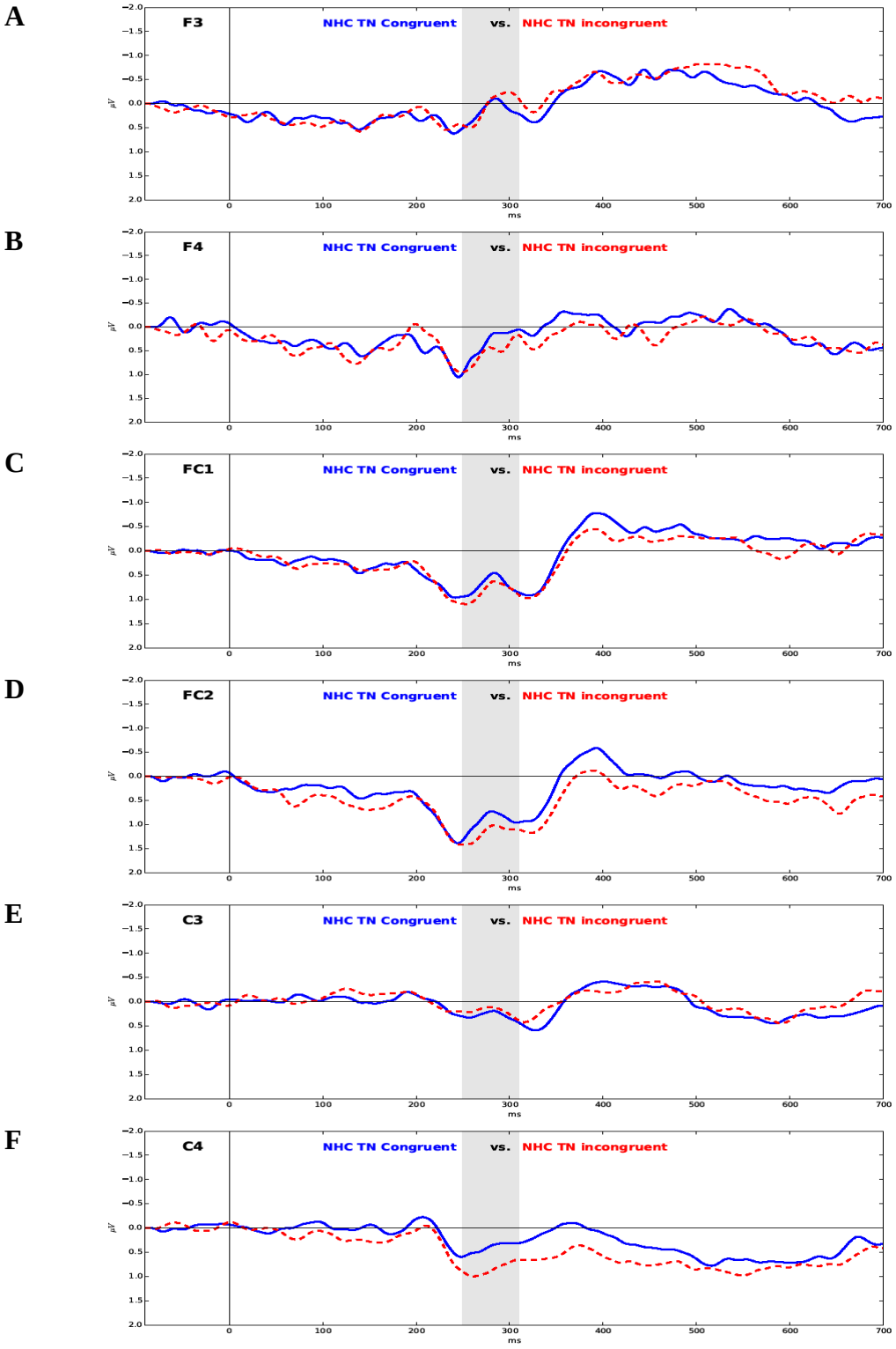


Figure 6.14. Grand average waveforms showing N2 μV (shaded grey) for TP congruent vs. incongruent DPT targets in the NHC condition at left compared to right frontal (panel A and B), frontocentral (panel C and D) and central sites (panels E and F)

With respect to prediction 10, analysis revealed no significant UHC x anger x congruency interaction ($p > 0.05$). Contrary to predictions 11 and 12, no N2 laterality difference was found between high and low anger participants or between RHCs, LHCs, and NHCs for TP targets independent of congruency. Inferential statistics for these results can be found in Table 6.18.

6.3.2.3.3. P3b ERP component

To explore whether compared to LHCs and NHCs, RHCs reduced P3b (reduced attentional bias towards threat) for congruent compared to incongruent TP word-pairs in high compared to low trait anger participants, a 3 (UHC condition; RHC, LHC, NHC) x 2 (Trait anger; high, low) x 2 (Congruency; congruent, incongruent) x 2 (Hemisphere; left, right) x 3 (Region; centroparietal, parietal, parietooccipital) mixed ANOVA was performed on P3b mean amplitudes for TP trials. Means and standard deviations of P3b amplitudes are presented for congruent and incongruent TP targets in relation to UHC for high and low anger participants in Table 6.19. *F* values and significance levels of this analysis are presented in Table 6.20.

In support of predictions 7, the ANOVA revealed no main effect of UHC, anger, congruency, site or hemisphere on P3b in response to TP word-pairs. Inferential information for these results can be found in Table 6.20. Contrary to predictions 8, 9, and 10, no P3b difference was found between high and low anger participants, or between RHCs compared to LHCs and NHCs, for TP congruent than incongruent targets in isolation or in relation to one another. Contrary to predictions 11 and 12, no P3b laterality difference was found between high and low anger participants or between RHCs, LHCs, and NHCs for TP targets. Inferential statistics for these results can be found in Table 6.20.

Table 6.19. Means and standard deviations of P3b amplitudes (μV) for TP targets in relation to the UHC, congruency and trait anger at left and right centroparietal, parietal and parietooccipital sites

UHC	TP congruence	Site	High Anger (n= 24)		Low anger (n=20)	
			M	*(SD)*	*M*	*(SD*
RHC	TP congruent	CP1	1.14	(1.51)	1.91	(1.56)
		CP2	1.07	(1.51)	2.23	(1.45)
		P3	0.86	(1.37)	0.79	(1.66)
		P4	0.83	(1.20)	1.27	(1.42)
		PO3	0.18	(1.50)	1.16	(2.11)
		PO4	0.90	(1.31)	0.77	(1.79)
	TP incongruent	CP1	1.01	(1.52)	1.33	(1.76)
		CP2	1.29	(1.30)	1.44	(1.70)
		P3	0.85	(1.14)	1.10	(1.45)
		P4	1.30	(1.36)	1.18	(1.28)
		PO3	0.85	(1.73)	1.07	(1.85)
		PO4	0.64	(1.65)	0.71	(1.71)
LHC	TP congruent	CP1	1.09	(2.10)	1.56	(1.66)
		CP2	1.74	(1.23)	1.89	(1.66)
		P3	0.59	(1.22)	0.93	(1.19)
		P4	0.97	(1.07)	1.14	(1.69)
		PO3	0.79	(1.97)	0.77	(1.26)
		PO4	0.82	(2.02)	0.75	(2.08)
	TP incongruent	CP1	1.10	(1.55)	1.59	(1.53)
		CP2	1.41	(1.37)	1.81	(1.81)
		P3	0.99	(1.31)	0.93	(1.61)
		P4	0.75	(1.10)	1.41	(1.54)
		PO3	0.84	(1.75)	0.55	(1.71)
		PO4	0.79	(1.79)	0.61	(1.51)
NHC	TP congruent	CP1	0.98	(1.32)	1.16	(1.91)
		CP2	0.99	(1.25)	1.50	(1.91)
		P3	0.90	(1.16)	1.07	(1.35)
		P4	1.25	(1.42)	1.13	(1.31)
		PO3	1.48	(1.65)	0.79	(2.26)
		PO4	0.90	(1.40)	0.51	(2.18)
	TP incongruent	CP1	1.20	(1.66)	1.31	(1.81)
		CP2	1.11	(1.24)	1.17	(1.77)
		P3	0.69	(1.17)	1.08	(1.66)
		P4	1.05	(1.47)	1.29	(1.49)
		PO3	0.88	(1.42)	1.25	(1.58)
		PO4	0.83	(1.54)	1.16	(2.67)

Table 6.20. Inferential statistics for the effect of UHC, congruence, region and hemisphere on P3b mean amplitudes (μV) to TP targets

	F	***p***
Trait anger	4.22	0.27
UHC	0.00	1.00
UHC × Trait anger	0.58	0.56
Congruency	0.01	0.91
Congruency × Trait anger	0.03	0.87
Site	3.49	0.06
Site × Trait anger	0.41	0.55
Hemisphere	0.43	0.51
Hemisphere × Trait anger	0.01	0.92
UHC × Congruency	0.08	0.92
UHC × Congruency × Trait anger	2.10	0.13
UHC × Site	2.65	0.06
UHC × Site × Trait anger	0.70	0.54
Congruency × Site	1.27	0.28
Congruency × Site × Trait anger	1.60	0.21
UHC × Congruency × Site	1.01	0.39
UHC × Congruency × Site × Trait anger	1.39	0.25
UHC × Hemisphere	0.55	0.58
UHC × Hemisphere × Trait anger	0.16	0.85
Congruency × Hemisphere	0.25	0.62
Congruency × Hemisphere × Trait anger	0.01	0.92
UHC × Congruency × Hemisphere	0.21	0.81
UHC × Congruency × Hemisphere × Trait anger	0.61	0.55
Site × Hemisphere	3.07	0.06
Site × Hemisphere × Trait anger	0.26	0.74
UHC × Site × Hemisphere	0.61	0.62
UHC × Site × Hemisphere × Trait anger	1.59	0.19
Congruency × Site × Hemisphere	0.28	0.74
Congruency × Site × Hemisphere × Trait anger	0.79	0.45
UHC × Congruency × Site × Hemisphere	1.58	0.19
UHC × Congruency × Site × Hemisphere × Trait anger	2.12	0.09

6.4. Discussion

Study 4 employed the DPT paradigm in combination with ERP indices of attentional bias, namely the P2, N2, and P3b to provide insight into whether the effects of the UHC method influenced evaluative (P2), inhibitory (N2) or motivational (P3b) aspects of attentional processing. Study 4 also explored the behavioural and neural effects of the UHC method in

relation to trait anger as trait anger is associated with greater relative left frontal brain activity (e.g. Harmon-Jones 2003) and increased attentional bias to threat (e.g. Smith and Waterman 2003). The findings from this study provide some of the first evidence that illustrates UHC influence changes in attentional bias to emotional words in the DPT paradigm. It also provides insight into the underlying mechanisms of these changes as measured by ERPs during the DPT. The results also provide some of the first evidence to illustrate how trait anger influences the effect of UHCs on attention bias to high arousing emotional words.

6.4.1. Summary of behavioural findings

Contrary to expectations (prediction 2) that regardless of UHC and trait anger, there would be an attentional bias towards threat compared to positive and neutral words, results showed attentional bias towards threat only when it was paired with equally high arousing positive words (in TP trials). In contrast, when threat and positive words were paired with low arousal neutral words (in TN and PN trials) there was avoidance of high arousing words regardless of their valence. The similarity of findings between TN and PN trials is consistent with evidence that emotional arousal and not valence influences attentional bias (e.g. Fischler and Bradley 2006; Herbert et al. 2008). However, when high arousing threat and positive words are presented together, attention towards threat words was prioritised, illustrating a true attentional bias to threat. This is in line with evidence that shows attentional bias to threat over positive words (e.g. Öhman 2005). However, it is important to note that this effect was opposite to that found in Chapter 5 where there was no attentional bias to threat presented with positive words but an attentional bias for threat when combined with neutral words. It is therefore possible that the EEG procedure in Study 4 induced anxiety which then produced a true attentional bias to threat. Although this is purely speculative without future exploration of EEG induced stress in relation to this study design.

It was also predicted that RHCs compared to LHCs and NHCs would reduce attentional bias to threat compared to positive and neutral words, in isolation of trait anger (prediction 3). This prediction was not supported in the present study. Contrary to predictions, the behavioural data provided no evidence that UHCs modulated attentional bias to emotion related words in isolation of trait anger. At first glance, this finding appears at odds with the assumption that increasing left frontal brain activity, via RHCs (Harmon-Jones 2006) will increase cognitive control over emotional responding (Davidson 2004) and therefore reduce attentional bias towards threat. However, it is important to note that if RHCs did increase cognitive control over attentional processing of threat words, this may have inhibited any measurable behavioural attentional bias.

It was also predicted that in isolation of UHCs, high compared to low trait anger participants, would show increased attentional bias to threat compared to positive and neutral words (prediction 4). Indeed attentional bias to emotional words was influenced by trait anger. However, contrary to predictions both high and low anger participants showed avoidance of positive compared to neutral words and vigilance towards threat compared to positive words. However, high and low anger participants did differ in response to threat compared to neutral words. High anger participants showed no attentional bias to threat, but low anger participants showed avoidance to threat. According to attentional bias theories, the threat-related avoidance suggested that low anger participants were able to counteract attentional bias to threat through strategic task-related efforts (Williams et al. 1996; Mathews and Mackintosh 1998). Indeed, this finding was consistent with previous evidence that showed avoidance to threat compared to neutral stimuli in low trait anger (e.g. van Honk et al. 2001).

It was also predicted that compared to LHCs and NHCs, RHCs will reduce attentional bias to threat compared to positive and neutral words and this effect will be larger in high than low anger participants (prediction 5). This prediction was not supported in the present study. Contrary to predictions, Study 4 also showed that when the UHC effects were explored in relation to trait anger, no UHC effects were found for PN or TP trials nor were any effects found RHCs or NHCs in TN trials. However, LHCs were found to increase attentional bias towards threat in high anger participants and increase threat avoidance in low anger participants when threat words were simultaneously presented with neutral words in the DPT. This finding diverged with trends that emerged in EST Study 2 (Chapter 3), where LHCs increased threat-related attentional bias in low anger participants. Furthermore, findings regarding the componential characteristics of UHC effects (Hypothesis 6) were inconclusive.

The discrepancy between findings in Study 2 (Chapter 3) and Study 4 may reflect the different research paradigms used in these studies. It has been suggested that the EST and DTP differ in terms of underlying processes, with the DPT measuring the allocation of attention at a later phase than the EST (Brosschot, de Ruiter, and Kindt 1999). While the EST measures responses to simultaneously presented stimuli (threat and colour), the DPT measures responses to targets presented after the offset of the threat stimuli. Furthermore, in the EST threat and colour information is integrated while in the DPT threat and target information is spatially and temporally separated. For this reason it is suggested that the EST and DPT measure different mechanisms (Brosschot, et al 1999). As such these methodological differences may explain the conflicting effects found for LHCs in low anger participants in Study 2 and 4. For example the differential patterns between tasks suggest that, following LHCs low anger participants had greater difficulty disengaging from threat when it was integrated with task relevant colour information, as in Study 2. In contrast,

findings from Study 4 indicate that following LHCs low anger participants successfully inhibited threat-related attention at the point of target onset reflecting avoidance at later processing stages.

6.4.2. Summary of ERP findings

In contrast to behavioural data and contrary to prediction 10, the ERP data from Study 4 showed no effect of the UHC method in relation to trait anger for threat compared to neutral targets. Furthermore, contrary to prediction 8, in isolation of UHCs, high anger participants showed significantly smaller parietal P3b for threat compared to neutral targets while no difference was found in low anger participants. According to the P3b literature (e.g. Duncan-Johnson and Donchin 1977; MacNamara et al. 2009) this finding suggests that high anger participants showed increased task difficulty and interference during the processing of threat congruent targets. Furthermore, as P3b attenuation was still evident after word offset, it can be assumed that attentional bias towards threat continued even after the word disappeared, possibly reflecting increased difficulty disengaging from threat. This finding is consistent with evidence that shows increased attentional bias towards threat in high anger participants (e.g. Smith and Waterman 2003). It is also consistent with evidence that shows anger-related attentional bias occur at later cognitive processing (e.g. van Honk et al. 2001)

With regards to prediction 9, Study 4 showed that in isolation of trait anger, there was a TN congruency effect for RHCs but not for LHCs or NHCs. Importantly, this showed that attention related P3b was modulated differently for RHCs compared to high anger. However, in contrast to prediction 9, RHCs increased parietooccipital P3b for threat compared to neutral targets. Evidence suggests that P3b is larger for target (congruent target) than non-target stimuli (word-pair) (e.g., Duncan-Johnson and Donchin 1977) and for emotionally

relevant compared to neutral stimuli (e.g., Johnston, Miller, and Burleson 1986; Keil et al. 2002; MacNamara et al. 2009). Therefore, this finding indicated that RHCs facilitated the detection of targets that replaced threat words without threat causing interference, as it did with high trait anger. This suggests that while RHCs increased the motivational relevance of threat (MacNamara et al. 2009) unlike high trait anger, RHCs did not reduce task performance (which would be indexed by reduced P3b for TN congruent targets). This assumption is consistent with evidence that RHCs increase approach motivated behaviour important for goal attainment (Harmon-Jones, 2006, Peterson et al., 2008). It also suggests that unlike high anger, RHCs also increased attentional control over threat interference to facilitate task processing. Therefore, this finding provides insight into the facilitated threat responding found in Study 1 (Chapter 3). It also provides support for the role of the left frontal brain in both emotion control (Davidson, 2004) and approach motivation (e.g., Davidson and Irwin 1999; Davidson et al. 2000)

In addition, Study 4 showed in line with predictions 8 and 10, that there was no effect of trait anger and no effect of UHCs in relation to trait anger on ERP modulations for PN congruency. However, contrary to prediction 9 independent of trait anger, an effect of UHCs was found in relation to PN congruency. While RHCs and LHCs were found not to influence a congruency effect, NHCs produced a trend for smaller frontocentral N2 for positive compared to neutral targets. According to previous evidence this suggested that there was greater conflict in the NHC than RHC and LHC conditions (e.g. Van Veen and Carter 2002). This assumption was supported by a trend where RHCs compared to both LHCs and NHCs reduced frontocentral N2 for positive incongruent targets only. According to Neural Efficiency Models (e.g. Van Veen and Carter 2002), the reduced N2 during the conflict induced by incongruent targets suggests that RHCs increased the neural efficiency of

processes used to shift attention from positive to neutral locations during task performance (Dennis and Chen 2007; Gray 2004; Gray, Braver, and Raichle 2002).

This assumption supports findings from Studies 1, 2, and 3 that indicated that RHCs, and therefore left frontal brain activity (Harmon-Jones 2006) increases the efficiency of attentional control mechanisms used to regulate emotion responding to improve task related processing. This is consistent with the view that left frontal brain activity is involved in increased cognitive control over emotional responding (Davidson, 2004) and approach-related, appetitive goals. (e.g. Davidson and Irwin 1999; Davidson et al. 2000). The finding also highlights that RHCs modulates not only attentional bias to threat, but also attention to highly arousing positive words. Therefore it highlights that RHCs may reduce attentional bias to any highly arousing stimuli. Possible implication of this will be discussed in the next section.

Study 4 also showed contrary to prediction 10 that for TP trials, ERPs were not modulated by the combined effect of UHC and trait anger in response to congruency. However in partial support of prediction 8, a P2 congruency effect was found for trait anger in isolation of UHCs. Though, while no P2 congruency effect was found for high anger participants, low anger participants showed a trend for larger P2 for threat compared to positive targets. According to previous evidence (Crowley and Colrain 2004; Eimer and Holmes 2007; Schapkin, Gusev and Kuhl 2000) this finding suggested that in low anger participants, threat was rapidly detected and evaluated to guide task motivated behaviour. This is consistent with previous evidence that has shown increased P2 for threat compared to positive stimuli (Carretie, Martin Loeches, Hinojosa, and Mercado 2001; Carretie, Mercado, Tapia, and

Hinojosa 2001). As no P2 response were found in relation to UHCs, (specifically RHCs) and high anger, this supports evidence that has shown that approach motivated attentional bias to threat is associated with the modulations of later cognitive processes only (e.g. Stewart et al. 2010). This also provides insight to future research to focus on later ERPs (N2, P3b, and NSW) when exploring the effects of the UHC method on attentional bias to threat.

Study 4 also showed in partial support for prediction 9, that independent of trait anger, N2 laterality was modulated by UHCs in response to TP congruency. For RHCs and LHCs, N2 was larger in the right hemisphere for TP incongruent than congruent targets, but in the left hemisphere there was no congruency effects. In the NHC condition, N2 was smaller for incongruent than congruent targets in the right hemisphere but again there was no congruency effect in the left hemisphere. According to previous evidence this finding suggests that RHCs and LHCs increased the recruitment of cognitive resources used to divert attentional bias away from threat words to targets replacing positive words (e.g. Dennis and Chen 2007; Gray 2004; Van Veen and Carter 2002). In contrast, NHCs may have been associated with reduced recruitment of inhibitory mechanisms in the right frontal brain, when shifts in attention from threat to positive locations were required, possibly reflecting the healthy sample of this study. This finding is consistent with fMRI evidence that has shown during the inhibition of dominant responses such as attentional bias towards threat, that increased activity the right frontal brain is found (e.g. Konishi, Nakajima, Uchida, Kikyo, Kameyama, and Miyashita 1999). The finding is also consistent with the view that the right frontal brain is particularly important in behavioural inhibition and vigilant attention to threat (e.g., Davidson and Irwin, 1999; Davidson, et al, 2000). A lack of difference between RHCs and LHCs at early cognitive processing stages indicates that methods with greater spatial

resolution are needed to explore which sectors of the PFC are differentially influenced by RHCs and LHCs to gain greater insight into these right hemisphere effects.

Similar to Study 2, the findings from the present Study 2 showed no effect of UHCs on laterality of ERP components. As such more appropriate methods are required in future research to explore the localisation of brain activity increases induced by the UHCs. This will be discussed in more depth in the following Chapter.

6.4.3. Further Considerations

This study provides novel data emphasizing the effectiveness of UHC in modifying attention bias to emotional words at both behavioural and electrophysiological levels. However, this study within this chapter is not without limitations and unresolved questions. For example, as early ERP components, namely P1 and N1 were not explored within this study; it cannot be said with any certainty that UHC did not also influence early perceptual attentional processes as well as later ones. Furthermore, as in previous studies within the Thesis (Studies 2, 3) effects were found for LHCs. As LHCs are known to increase contralateral brain activity and right frontal brain activity is associated with withdrawal motivation and negative effect (e.g. Sutton and Davidson 1997) future research should explore UHCs in relation to individual difference characteristics such as trait anger. As such further work is needed in this area. This is discussed in detail in the following Chapter.

CHAPTER 7

GENERAL DISCUSSION

7.1 Introduction

This chapter will integrate the findings from the studies presented in this Thesis, discuss their implications, and identify areas for future research. The first section of this Chapter will consider the role of the UHC method and trait anger in the modulation of attentional bias to emotion related words. In the second section, the strengths and limitations of the four studies within this Thesis are discussed. The third section then discusses the implications of the findings in terms of theories of frontal brain asymmetry and in terms of informing interventions to reduce threat-related attentional biases to improve task performance. Finally, areas for future research that will enhance the understanding of the UHC method for modulating attentional bias to emotion related stimuli are discussed.

7.2 Summary of Main Findings

The findings of this Thesis serve three main purposes: 1) They integrate the UHC, attentional bias and frontal brain asymmetry literature, 2) They confirm previous evidence regarding the role of greater left frontal brain activity, approach motivation and anger on attentional bias to emotion related words and 3) They provide novel information on the usefulness of the UHC method in modulating attentional bias to emotion-related words.

Anger-related behaviours are contributory factors for problems in interpersonal relationships, health and society (Gardener and Moore 2008). In addition, attentional bias to threat plays a

fundamental role in the causation and maintenance of anger (Gardener and Moore 2008). Identifying the underlying mechanisms of anger-related attentional bias enables a better understanding of anger generation and could have practical implication for informing efficient anger reduction interventions in people susceptible to anger. Furthermore, anger as an approach motivated negative emotion, has been associated with greater left frontal brain asymmetry (Harmon-Jones 2003). However, greater left frontal brain activity has also been associated with increased cognitive control of emotional responding (attentional bias to threat). Therefore, a lack of clarity remains about the role of the left frontal brain in attentional bias to threat. For instance, attentional bias to threat has been positively associated with both greater left (e.g. d'Alfonso et al. 2000) and right frontal brain activity (Gray 1994). These inconsistencies left questions regarding the role of frontal brain asymmetry in attentional bias to threat and its interaction with anger. The remainder of this section will discuss the main findings of this Thesis in relation to the role of UHCs in the modulation of anger-related attentional bias in order to provide insight into these inconsistencies. Overall, the findings presented in this Thesis have provided novel information about the effectiveness of the UHC method to modulate attentional bias to emotion related words across different attentional paradigms. They have also highlighted the influence of individual differences that trait anger has on the effect of the UHC method in attention modification.

Independent of trait anger, RHCs appear to motivate attention towards threat as indexed by enhanced P3b (Studies 2 and 4) Chapter 4 and 6) but subsequently increased threat-related avoidance at a behavioural level (Studies 1 and 3). This increased avoidance following RHCs appeared to be a result of increased efficiency of left frontal neural mechanisms used to inhibit threat conflict, as indexed by reduced NSW for threat compared to neutral words

(Study 2). It was therefore assumed that RHCs had increased the ability to disengage attention from threat compared to neural words (all studies) and for threat compared to positive words (Studies 3 and 4) which facilitated task performance. In summary, the findings in this Thesis provide some of the first evidence that RHCs reduce attentional bias to threat compared to neutral words. Furthermore they show that the underlying mechanisms of this effect was linked to an increased neural efficiency of later control mechanisms at left frontal sites (Study 2) and increased motivational processing at centroparietal-parietooccipital sites (Study 4). The Thesis also provides some of the first evidence that RHCs modulate attentional bias to high arousal words in general. RHCs increased the recruitment of early control mechanisms in the right hemisphere (indexed by increased N2) to shift attention from threat to positive locations and increased the neural efficiency of frontal-frontocentral control mechanisms to shift attention from positive to neutral locations as evidenced in Study 4.

These findings support motivational models of frontal brain asymmetry (e.g. Davidson 1995) in that increased approach motivation will guide and motivate goal relevant behaviour (for a review see, Harmon-Jones et al. 2010). These findings somewhat contradict evidence that shows RHCs increase aggression (Peterson et al. 2008) as it would be expected that as attentional bias to threat predicts aggression and RHC reduced attentional bias towards threat this should subsequently reduce aggression. However, in Petersons study (2008), the goal relevant behaviour was to approach threat in an attempt to remove the stressor where as in the present research goal relevant behaviour was task oriented target responses. As such it can be postulated that, the effects of RHC increased approach motivation will be dependent on the goal at hand.

Interestingly, independent of trait anger, the findings from this Thesis illustrate that LHCs significantly increased difficulty disengaging from threat compared to neutral words and increased avoidance for positive compared to neutral words (Study 3). The ERP findings revealed trends where it appears that LHC increase attentional bias at all cognitive processing stages. First, LHCs increased recruitment and reduced the efficiency of early right frontal-frontocentral control mechanisms used to inhibit threat-related interference (Study 2). This was indexed by enhanced N2 for threat compared to neutral words. Second, LHCs increased motivational processing of threat compared to neutral words at left parietooccipital sites, which was assumed to increase task difficulty (Study 2). This was indexed by reduced P3b. Finally LHCs reduced the recruitment of right frontocentral cognitive control mechanisms to resolve threat conflict at later inhibitory stages, indexed by reduced NSW (Study 2).

Independent of UHCs, high compared to low anger participants showed increased motivational processing at parietal sites for threat compared to neutral words which was assumed to reflect increased difficulty shifting attention from threat to neutral locations (Study 4). This was indexed by smaller P3b for threat compared to neutral words. This may indicate that the attention of high anger participants was drawn to high arousal rather than threat per se, as no effects were found for threat when compared to equally high arousing positive words. Nevertheless, following LHCs high anger participants showed increased motivational processing of threat at centroparietal sites (increased P3b) and reduced recruitment of frontocentral inhibitory mechanisms (NSW) to resolve threat conflict (Study 2). This effect was further illustrated in the DPT behavioural data, as an increased threat-related attentional bias (Study 4). As LHCs are known to increase negative affect (Schiff and Lammon 1994), this suggests that increased negative affect in high anger participants further increases the motivational relevance of threat and reduces the ability to control threat

processing in favour of processing competing task relevant information. Given that RHCs are known to increase goal relevant approach motivation (Harmon-Jones 2006) it is unclear as to why no effects of RHC were found in relation to high anger. It is possible that a ceiling effect occurred in these participants rendering the RHC as ineffective in reducing attentional bias to threat in these participants. Further research is required that focuses on the effect of RHCs at later cognitive control processing stages in relation to high anger in order to investigate this further.

In contrast, in isolation of UHCs low compared to high anger participants showed threat avoidance when attention from threat to neutral locations was required (Chapter 6). Furthermore, low anger participants showed early rapid detection and increased evaluation of threat compared to positive words (Chapter 6). Collectively this may suggest that after an initial normative vigilant attention to threat and increased threat evaluation that attention is then subsequently directed to opposite locations to threat stimuli. Furthermore, it also suggests that in low anger participants attention to threat is prioritised over equally high arousing positive words, demonstrating a true attentional bias to threat. However, a lack of behavioural interference suggests that this early attentional bias to threat did not in fact reduce task performance. However, following LHCs low anger participants showed an increased threat-related attentional interference at a behavioural level (Chapter 4) but following RHCs they showed a trend for increased approach motivated task relevant processing at centroparietal regions for threat compared to neutral words, as indexed by enhanced P3b (Chapter 4). This highlights the effectiveness of UHCs to modulate attentional processing of emotion related words in low anger individuals.

In summary the findings of this Thesis provide some of the first to show that RHCs reduce attentional bias to threat in isolation of trait anger and in individuals with low trait anger. However, findings also show that RHCs do not modify attentional bias to threat in high anger individuals. In contrast, the Thesis also provides novel evidence that the LHC increases attentional bias to threat and this reduces task relevant processing, regardless of trait anger level. As the above paragraphs summarise the broad main findings of the body of work presented in this Thesis, Section 7.4 will discuss these findings in relation to both the theoretical and practical implications of this work. The next section will evaluate the strengths and limitations of the Thesis overall.

7.3 Evaluation of the Research

7.3.1 Strengths of Research

The aim of the studies conducted in this Thesis was to provide new information about the effects of the UHC method (Schiff and Lamon 1994) in the modulation of attentional bias to emotion-related words and how trait anger influences its effects. The studies reported in this Thesis have a number of significant strengths. First, it is the first research to employ the use of the UHC method to modulate attentional components of emotion regulation. Previous research employing this method has explored its effects on later response focused components of emotion regulation such as emotion expression and has provided no insight into how the UHC method modulates more automatic emotion regulation components such as attentional deployment (e.g. Schiff and Lamon 1994, Harmon-Jones 2006; Peterson et al. 2008).

Second, only a handful of studies have explored attentional bias to threat in relation to high anger (e.g. Eckhardt and Cohen 1997; Smith and Waterman 2003; van Honk, Tuiten, de Hann, van den Hout, and Stam 2001; van Honk, Tuiten, van den Hout et al. 2001). The correlational nature of previous research has provided no insight into the causal relationship between anger and attentional bias to threat. The research presented in this Thesis offers novel information about causal relationship between the increased activation of anger-related networks and attentional bias to emotion related words.

Third, a number of different methodologies were employed ranging from EST analysis of attentional bias to threat (Studies 1 and 2), DPT analysis of attentional bias to emotion related words (Studies 3 and 4), and the analysis of ERP measures of cognitive components of attentional bias to emotion related words (Studies 2 and 4) and self-reported measures of trait anger (Studies 2, 3 and 4). This has allowed for the measurement of attentional bias modification across diverse response and neural domains. In doing so it has provided novel insight and a comprehensive assessment of the effects of the UHC method on attentional bias to emotion related words both in relation to and independent of trait anger.

Finally, two of the studies in this Thesis explored the effect of the UHC method on attentional bias to high arousing emotion related words presented with low arousal neutral words (Studies 3 and 4). Previous research has indicated that using both high arousing threat and positive stimuli in addition to neutral stimuli allows for a comprehensive examination of whether attentional biases reflects valence or arousal and ensures that valence and arousal are not confounded (e.g. Kanske and Kotz 2007). Exploring attentional bias to high arousing emotion related words when these were presented with low arousal neutral words provided an

index of arousal. In contrast, exploring attentional bias towards high arousal threat words when they were presented with equally high arousing positive words provided an index of true attentional bias to threat. This exploration provided novel insight into whether the UHC method modulated arousal or valence dimensions of attentional bias.

Overall, the studies presented in this Thesis provide a comprehensive account of how the UHC method and the effects of anger modulates valence and arousal dimensions of attentional bias to threat across different attentional bias paradigms. The ERP findings also indicate that the UHC method reliably modulates the neural processes that underpin attentional bias to threat.

7.3.2 Limitations of Research

This research has a number of potential limitations which should also be considered. First, measures of state and trait anxiety were not controlled for in the studies presented in this Thesis. There is consistent evidence to show that there is a positive association between negative affect syndromes and attentional bias to threat (Bar-Haim et al. 2007). In addition, EEG recording involves mildly unpleasant preparation procedures that can induce anxious states in the high trait anxious individuals which can cause ceiling effects (Blackhart, Kline, Donohue, Larowe, and Joiner 2002). It is not possible to ascertain the impact that individual differences in anxiety may have had on the findings presented in this Thesis. Gaining measures of baseline anxiety levels and pre and post EEG preparation would control for potential confounds in future studies. However, given the lack of research exploring attentional bias in relation to anger and anger-related networks, it was empirically and

theoretically important that the research remained focused on the role of the UHC in relation to anger. Furthermore, the addition of further constructs was limited by issues of statistical power.

Second, the studies presented in this Thesis did not explore whether UHCs modulated phasic contralateral frontal brain activity. For this reason, inferences made about the UHC effects on frontal brain asymmetry are somewhat speculative. Previous research has indeed shown the RHCs reliably increase activation of central and frontal regions of the contralateral hemisphere (Harmon-Jones 2006; Peterson et al. 2008). In contrast, evidence for the contralateral activation of LHCs is inconsistent with some evidence showing that LHCs produce greater right than left mid-frontal, anterior-temporal and parietal activity (Harmon-Jones 2006) and others showing increase activation of left posterior regions (Peterson et al. 2008). Recording EEG measures of Mu oscillations (8-12Hz; μ rhythm) during UHCs would allow more direct inferences to be made about the effects of the UHC method on activation of the contralateral motor and frontal cortex. This method has been successfully implemented in past research exploring the influence of UHCs in manipulating frontal brain asymmetry (e.g. Harmon Jones 2006). Furthermore, employing the use of methods with both a high temporal and spatial resolution, such as Magnetoencephalography (Sekihara, Nagarajan, Poeppel, and Marantz 2002) to explore gamma oscillations in relation to the UHC may provide greater insight into the localisation of the UHC effect.

Third, participants were all right-handed as measured by the Edinburgh handedness laterality quotient (Williams 1986) in order to reduce variance in the EEG data. Therefore, hand-contractions in the non-dominant left hand may have resulted in greater effort and muscle

fatigue being recruited during LHCs than in RHCs. As such this could account for the greater attentional bias to threat and presumably negative affect shown for LHCs. Measures of negative affect or perceived fatigue pre and post hand-contraction could control for this in future studies.

Fourth, all of the studies in this Thesis included a greater number of female than male participants. Language functions are known to be represented more bilaterally in females (Gur, Alsop, Glahn, and Petty et al. 2000), which in turn is suggested to interfere with visuospatial functions in the right hemisphere (Sommer, Aleman, Bouma and Kahn 2004). Therefore, it is possible that the effect of the UHC method on attentional bias to emotion related words would be stronger in males. The predominantly female sample may have reduced the size of any effects observed.

Finally, ERP data (Chapters 4 and 6) focused on later cognitive ERP components of attention and as such, the findings provide no insight into the effects of early attentional modifications of the UHC method. However, the focus on later ERPs was guided by evidence suggesting that anger-related attentional bias is confined to later cognitive processes (e.g. Smith and Waterman 2003; van Honk et al. 2001; Stewart et al. 2010). Nevertheless, the exploration of the effects of the UHC method on early attentional ERP components would have further strengthened this assumption.

7.4 . Implications

7.4.1 Theoretical Implications

The findings presented in this Thesis have important theoretical implications for the role that differential patterns of frontal brain asymmetry play in regulating attentional components of emotion. According to Diathesis Models (e.g. Davidson 1992, 1994), individual differences in frontal brain asymmetry should result in different responses to an elicitor of negative emotion, such as threat.

The research presented in this Thesis has provided causal evidence that increased left frontal brain activity, via RHCs (Harmon-Jones 2006), increased approach motivated goal relevant behaviours in the face of high arousing stimuli. Therefore, this Thesis has provided support for the role of the left frontal brain in controlling and regulating emotional responding (Davidson et al. 2004) and for motivational models of frontal brain asymmetry that posit a positive association between left frontal brain activity and approach motivation (e.g. Davidson and Irwin 1999; Davidson et al. 2000). In doing so, data from the studies in this Thesis also provide support for Gray's (1994) approach motivated BAS (also linked to greater left frontal brain activity (see, Harmon-Jones et al. 2010), motivates behaviours towards goals that promote reward and non-punishment. Therefore, it could be assumed greater left frontal brain activity, via RHCs; increased approach motivated attentional processing of goal relevant information even in the presence of negatively valence stimuli. This supports previous UHC research that has also shown RHCs increased approach motivation and aggression (Harmon-Jones 2006, Peterson et al. 2008). The present research also provides important insight to show that the increased aggression found for RHCs in previous research (Peterson et al. 2008) is caused by increasing approach motivated attention

towards threat to engage in goal relevant behaviours, where in the case of aggression these would be to remove the stressor.

In addition, the findings of this research show, although somewhat tentatively, that LHCs, and presumably increased right frontal brain activity (Harmon-Jones 2006) increase behavioural inhibition and increase attentional bias to threat. This finding is consistent with evidence that has shown that greater right frontal brain activation relates to deficits in the approach system and in reward-related responding (Henriques and Davidson 2000). However, according to Gray's (1994) Motivational Theory, the withdrawal related behavioural inhibition system (BIS) inhibits behaviour and increases attention towards threat, during non-reward and threat induced fear. In line with this theory, it is possible that LHCs increased activity of the BIS in individuals which increased their attentional bias towards threat. However, it is important to note that while BAS has been consistently associated with greater left frontal brain activity, the association between BIS and greater relative right frontal brain activity has been more elusive (for a review see Harmon-Jones, Gable, and Peterson 2010). Therefore, assumptions about the positive association between right frontal brain activity via LHC and withdrawal related BIS are purely speculative at present and further research is needed to support this claim.

In contrast, there has been more consistent support for the role of right frontal brain activity in relation to the valence hypothesis that posits that the right PFC is associated with negative affect (e.g. Tomarken, et al. 1992; Davidson 1984, 1998). Indeed, the findings presented in this Thesis support previous evidence that LHCs increase negative affect as well as bias perceptions and judgments negatively (Schiff and Lamon 1994). The findings are also in line

with the view that greater right frontal brain activity (Perez-Edgar et al. 2013) and negative affect is positively associated with attentional bias towards threat (Bar-Haim et al. 2007).

Therefore, it could be assumed from the evidence presented within this Thesis, that in the presence of a threat, RHCs increase activity of left frontal networks associated with approach motivation and LHCs increase activity of right frontal networks associated with withdrawal motivated negative affect. However, as valence and motivation are confounded in the LHC condition (negative stimuli often results in withdrawal behaviours), further investigations that allow valence and motivation to be studied independently are needed to identify whether LHC modulated withdrawal motivation or negative affect. Furthermore, it is essential that the current findings are replicated and extended before more in depth theoretical underpinnings can be provided for the UHC effects. However, the findings do suggest that, in accordance with previous literature (see Harmon-Jones 2004), in the presence of high arousal stimuli, left frontal brain activity is more psychologically beneficial than right frontal brain activity (Harmon-Jones 2003).

7.4.2 Attention Modification for Interventions and Research

The findings presented in this Thesis have also provided evidence on the effectiveness of RHCs in reducing not only attentional bias to threat per se, but also reducing attentional bias to highly arousing positive words. However, contrary to predictions that RHCs may be useful in the reduction of approach motivated attention in relation to anger, the findings in this Thesis suggest that RHCs would be ineffective in relation to high trait anger. In fact, they suggest that if an individual's goal was to remove a stressor then RHCs would increase the propensity to engage in goal oriented attention that may increase aggressive behaviour. This

is consistent with previous evidence that has shown RHCs increase aggression (Peterson et al. 2008). As such RHCs would not reduce aggression in individuals predisposed to high anger.

However, current findings do suggest that independent of trait anger, RHCs reduce attentional bias to high arousing emotional stimuli and facilitate goal orientated task responding. As such, this has important implications for improving goal performance in the presence of a threat in healthy individuals. The processing of personally relevant threat has been consistently shown to reduce the amount of attention available for carrying out task relevant processing (Derakshan and Eysenck 2009). According to the Attentional Control Theory (Eysenck, Derakshan, Santos, and Calvo 2007), threat impairs the efficiency of top down processes involved in the inhibition of attention to task-irrelevant threat. Given that RHCs have been shown to increase the efficiency of attentional control mechanisms this method could then be used to improve task performance in healthy individuals.

The findings presented in this Thesis also have important implications for future research in the area of attentional bias. They have shown that independent of individual differences in trait anger, LHCs increased attentional bias to threat and that this was possibly a result of increased negative affect (Schiff and Lamon 1994). More importantly, this effect was found across both the EST and DPT paradigms. As research has consistently shown that negative affect is associated with attentional bias to threat (Bar-Haim et al. 2007) the findings indicate that this method may be successfully employed in attentional bias research in healthy samples to induce attentional patterns similar to that of clinical anxious populations, to inform theory and attentional bias modification interventions.

7.5 . Future Research

The research presented in this Thesis suggests some interesting avenues for future research. The present findings illustrated that RHCs were ineffective at reducing attentional bias to threat in high trait anger participants. However, the findings in this research provide no insight into whether RHCs will modulate attentional bias during anger inducing situations. It is possible that high state anger may need to be induced for an effect to be seen in high trait anger individuals. This assumption is in line with the view of the Diathesis Stress Model (Davidson 1992) where according to this, RHCs would not be sufficient to induce a change in affective behaviour. Instead, modulations of threat-related attentional bias would only be expected in response to RHCs if an individual was predisposed to high anger and in an anger inducing situation.

Furthermore, given that the findings presented within this Thesis suggest LHCs increased negative affect and attentional bias towards threat, future research should explore the effect of UHCs in relation to trait and state negative affect and anxiety. It is possible that increasing left frontal brain activity via RHCs may provide an effective attention bias modification method to reduce attentional bias in relation to negative affect. At present attentional modification programmes that aim to reduce anxiety focus on the use of computerised attention tasks such as the DPT. The purpose of these tasks is to systematically train attention away from threat and improve the efficiency of task performance (O'Toole and Dennis 2012; Eldar and Bar-Haim 2010). Similar to the UHC method, these programmes influence relatively late cognitive processes (Eldar and Bar-Haim 2010). Therefore, if RHCs can be shown to increase task relevant processing and avoidance to threat in anxious individuals, the

UHC method could be used in combination with computerised tasks or provide a less costly alternative to it.

Furthermore, as the current research does not provide information on the role of RHCs in preconscious processing of threat information, future research should employ shorter stimuli presentation durations (<500ms) in masked EST and DPT paradigms. This would provide an opportunity to explore whether RHCs also induce approach motivated task relevant processing and LHCs induce attentional bias to threat even when threat is prevented from reaching conscious awareness. This would also provide insight into whether the UHC also modulates early, automatic, attentional processing of threat as well as providing further information on the time course of the UHC effects.

In this Thesis the effects of the UHC method on the modification of threat-related attentional bias was studied using word stimuli with either a negative, positive or neutral word valence. However, the use of words as stimuli has been suggested to raise methodological issues relating to confounding stimulus threat value and subjective fluency (Mogg and Bradley 1999). For example, in high anger participants threat words (e.g. *kill, punch*) are likely to be used more frequently than in low anger participants. This may in turn mediate the effects of attentional bias to threat due to priming effects. However, evidence has shown that interventions reduce affect induced attentional biases (e.g. Mathews, Mogg, Kentish, and Eysenck 1995) suggesting that word frequency may not be a critical confound. Another issue with word stimuli is that words lack ecological validity and as such are somewhat inadequate for evoking a true emotional response (e.g. the word `*punch*' is not very threatening for most people). Therefore, research using word stimuli may provide limited insight into attentional

biases for threat. In contrast, emotional faces may provide a more ecologically valid threat cue in future research and in doing so may provide a truer reflection of the effects of the UHC method.

The avenues for future research discussed above will extend the novel findings generated in this Thesis and allow for the development of the UHC method as an attentional modification tool in future research to further explore the role of frontal brain asymmetry and as an alternative method to computerised training in attention modification programmes.

7.6. Final Remarks

The research within this Thesis set out to explore whether UHCs could modulate attentional components of emotion regulation. This research was important as previous research utilising the UHC method had only explored its effects on experiential and behavioural components of emotion regulation (e.g. Harmon-Jones 2006). Furthermore, a paucity of previous research exploring attentional bias in relation to approach motivated anger and left frontal brain activity has left knowledge gaps both theoretically and empirically about the functional role of the left frontal brain in attentional bias to threat. This body of research aimed to bridge this knowledge gap and provide novel insight into the role of induced increases in approach motivation and greater relative left frontal brain activity via the RHC on attentional bias to emotion-related words. In doing so it attempted to provide further insight into the hemispheric specialisation of cognitive components of emotion regulation.

The four studies within this Thesis utilised a mixture of EST and DPT paradigms which were quantified through RTs and ERPs in isolation and in relation to trait anger. In doing so, the

studies within this Thesis demonstrated that UHCs modulated attentional bias to emotion related words across paradigms at either a behavioural of neural level. Specifically, the Thesis produced novel evidence that RHCs reduce threat-related attentional bias and enhanced task-related processing in isolation of trait anger, albeit with some inconsistencies. The findings also provided novel evidence that LHCs increased attentional bias to threat words and increased avoidance to positive words in low trait anger participants.

Collectively, this body of research is theoretically important as it provides support for influential models of frontal brain asymmetry. The reduced threat-related attentional bias and enhanced task processing found following RHCs provided support for models that claim the left PFC is implemented in approach motivation (e.g. Harmon-Jones 2004) and cognitive control (e.g. Davidson 2003; 2004). In contrast, the increased attentional bias to threat words and increased avoidance to positive words found in low trait anger participants, following LHCs provided support for models that posit the right PFC is implemented in negatively valenced emotions (e.g. Davidson, 1984), attentional bias to threat (e.g. Gray 1987) and withdrawal motivation (Davidson 1998).

These finding also have practical implications in that this body of research provides novel evidence that the UHC method can reliably induce attentional changes across paradigms and in doing so highlights the effectiveness of UHCs to explore hemispheric specialisation of cognitive components of emotion regulation. Therefore, this Thesis opens up many exciting avenues for further research in the field of frontal brain asymmetry.

REFERENCES

Abdu, R., Shinar, D., and Merian, A. (2012) 'Situational anger and driving'. *Transportation Research Part F: Traffic Psychology and Behaviour* 15, 575–580

Ahern, G. L., and Schwartz, G. E. (1985) 'Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis'. *Neuropsychologia* 23 (6), 745-755

Alfano, K. M., and Cimino, C. R. (2008) 'Alteration of expected hemispheric asymmetries: Valence and arousal effects in neuropsychological models of emotion'. *Brain and Cognition* 66 (3), 213-220

Algom, D., Chajut, E., and Lev, S. (2004) 'A rational look at the emotional Stroop phenomenon: A generic slowdown, not a Stroop effect'. *Journal of Experimental Psychology: General* 133 (3), 323-338

American Psychiatric Association (2000) *Diagnostic and statistical manual of mental disorders* DSM-IV-TR. Washington, DC: American Psychiatric Association

Amir, N., Beard, C., Burns, M., and Bomyea, J. (2009) 'Attention modification program in individuals with generalized anxiety disorder'. *Journal of Abnormal Psychology* 118, 28–33

Amir, N., Beard, C., Taylor, C. T., Klumpp, H., Elias, J., and Burns, M. (2009) 'Attention training in individuals with generalized social phobia: a randomized controlled trial'. *Journal of Consulting and Clinical Psychology* 77 (5), 961–973

Amir, N., Elias, J., Klumpp, H., and Przeworski, A. (2003) 'Attentional bias to threat in social phobia: Facilitated processing of threat or difficulty disengaging attention from threat?' *Behaviour Research and Therapy* 41 (11), 1325–1335

Amodio, D. M., Master, S. L., Yee, C. M., and Taylor, S. E. (2008) 'Neurocognitive components of the behavioral inhibition and activation systems: Implications for theories of self-regulation'. *Psychophysiology* 45 (1), 11–19

Anderson, C. A., and Bushman, B. J. (2002) 'The general aggression model: An integrated social-cognitive model of human aggression'. *Annual Review of Psychology* 53

Anton, S. D., and Miller, P. M. (2005) 'Do negative emotions predict alcohol consumption, saturated fat intake, and physical activity in older adults?' *Behavior Modification*, 29 (4), 677–688

Banich, M. T. (2009) 'Executive function: The search for an integrated account'. *Current Directions in Psychological Science*, 18(2), 89–94

Barbour, K. A., Eckhardt, C. I., Davison, G. C., and Kassinove, H. (1998) 'The experience and expression of anger in maritally violent and maritally discordant-nonviolent men'. *Behavior Therapy* 29 (2), 173-191

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., and van IJzendoorn, M. H. (2007) 'Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study'. *Psychological Bulletin* 133 (1), 1–24

Bar-Haim, Y., Lamy, D., and Glickman, S. (2005) 'Attentional bias in anxiety: A behavioral and ERP study'. *Brain Cognition* 59 (1), 11–22

Barlow, D. H. (2002) *Anxiety and its disorders: the nature and treatment of anxiety and panic*. New York: Guilford Press

Barlow, D. H., Allen, L. B., and Choate, M. L. (2004) 'Toward a unified treatment for emotional disorders'. *Behavior Therapy* 35 (3), 205-230

Barrett, L. F. (2006) 'Solving the emotion paradox: Categorization and the experience of emotion'. *Personality and Social Psychology Review* 10 (1), 20–46

Beck, A. T. (1976) *Cognitive therapy and the emotional disorders*. New York: International Universities Press

Beck, A. T., and Clark, D. A. (1997) 'An information processing model of anxiety: Automatic and strategic processes'. *Behaviour Research and Therapy* 35 (1), 49−58

Benazzi, F. (2003) 'Major depressive disorder with anger: A bipolar spectrum disorder?' *Psychotherapy and Psychosomatics* 72 (6), 300-306

Berkman, E. T., and Lieberman, M. D. (2010) 'Approaching the bad and avoiding the good: Lateral prefrontal cortical asymmetry distinguishes between action and valence'. *Journal of Cognitive Neuroscience* 22 (9), 1970–1979

Berkowitz, L. (1990) 'On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis'. *American Psychologist* 45 (4), 494-503

Berkowitz, L. (1993) *Aggression: Its Causes, Consequences, and Control*. New York: McGraw-Hill

Berkowitz, L., and Harmon-Jones, E. (2004) 'Toward an understanding of the determinants of anger'. *Emotion* 4 (2), 107–130

Bertsch, K., Böhnke, R., Kruk, M. R., and Naumann, E. (2009) 'Influence of aggression on information processing in the emotional stroop task–an event-related potential study'. *Frontiers in Behavioural Neuroscience* 3 (28), 1-20

Bertsch, K., Böhnke, R., Kruk, M. R., Richter, S., and Naumann, E. (2011) 'Exogenous cortisol facilitates responses to social threat under high provocation'. *Hormones and Behavior* 59 (4), 428–434

Bettencourt, B. A., Talley, A., Benjamin, A. J., and Valentine, J. (2006) 'Personality and aggressive behaviour under provoking and neutral conditions: A meta-analytic review'. *Psychological Bulletin* 132 (5), 751–777

Bishop, S. J. (2007) 'Neurocognitive mechanisms of anxiety: an integrative account'. *Trends in Cognitive Sciences* 11 (7), 307–316

Bishop, S. J. (2008) 'Neural mechanisms underlying selective attention to threat'. *Annals of the New York Academy of Science* 1129, 141–152

Bishop, S. J. (2009) 'Trait anxiety and impoverished prefrontal control of attention'. *Nature Neuroscience* 12, 92–98

Blackhart, G. C., Kline, J. P., Donohue, K. F., Larowe, S. D., and Joiner, T. E. (2002) 'AVective responses to EEG preparation and their link to resting anterior EEG Symmetry'. *Personality and Individual Differences* 32, 167–174

Blackhart, G. C., Minnix, J. A., and Kline, J. P. (2006) 'Can EEG asymmetry patterns predict future development of anxiety and depression? A preliminary study'. *Biological Psychology* 72 (2), 46-50

Botvinick, M., Braver, T. S., Barch, D. M., Carter, C. S., and Cohen, J. D. (2001) 'Conflict monitoring and cognitive control'. *Psychological Review* 108 (3), 624–652

Botvinick, M, M., Cohen, J. D., Carter, C. D. (2004) 'Conflict monitoring and anterior cingulate cortex: an update'. *Trends in Cognitive Science* 8 (12), 539–46

Bower, G. H. (1981) 'Mood and memory'. *American Psychologist* 36 (2), 129-148

Bradley, M. M., and Lang, P.J. (1999) *Affective norms for English words (ANEW): Stimuli, instruction manual and affective ratings. Technical report* C-1, Gainesville, FL. The Center for Research in Psychophysiology, University of Florida

Bradley, B. P., Mogg, K., White, J., Groom, C., and de Bono, J. (1999) 'Attentional bias for emotional faces in generalised anxiety disorder'. *British Journal of Clinical Psychology* 38 (3), 267–278

Bradley, B. P., Mogg, K., Falla, S. J., and Hamilton, L. R. (1998) 'Attentional bias for threatening facial expressions in anxiety: Manipulation of stimulus duration'. *Cognition and Emotion* 12 (6), 737–753

Bradley, B. P., Mogg, K., Millar, N., and White, J. (1995) 'Selective processing of negative information: Effects of clinical anxiety, concurrent depression, and awareness'. *Journal of Abnormal Psychology* 104 (3), 532−536

Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., Snyder, A. (2001) 'Anterior cingulate cortex and response conflict: Effects of frequency, inhibition and errors'. *Cerebral Cortex* 11 (9), 825–836

Brosschot, J. F., de Ruiter, C., and Kindt, M. (1999) ‘Processing bias in anxious subjects and repressors, measured by emotional Stroop interference and attentional allocation’. *Personality and Individual Differences* 26 (5), 777-793

Browning, M., Holmes, E. A., Murphy, S. E., Goodwin, G. M., and Harmer, C. J. (2010) 'Lateral prefrontal cortex mediates the cognitive modification of attentional bias'. *Biological Psychiatry* 67 (10), 919–925

Burgess, A. P. (2012) 'Towards a unified understanding of event-related changes in the EEG: the firefly model of synchronization through cross-frequency phase modulation'. *PLOS ONE*, 7 (9) :e45630 10.1371/journal.pone.0045630

Canli, T., Desmond, J. E., Zhao, Z., Glover, G., and Gabrieli, J. D. (1998) ‘Hemispheric asymmetry for emotional stimuli detected with fMRI’. *Neuroreport* 9 (1), 3233-3239

Carretié, L., Hinojosa, J.A., Martin-Loeches, M., Mercado, F., and Tapia, M. (2004) 'Automatic attention to emotional stimuli: neural correlates'. *Human Brain Mapping* 22 (4), 290–299

Carretie, L., Mercado, F., Tapia, M., and Hinojosa, J. A. (2001) 'Emotion, attention, and the ‘negativity bias’, studied through event-related potentials'. *International Journal of Psychophysiology* 41 (1), 75–85

Carver, C. S. (2004) ‘Negative affects deriving from the behavioral approach system’. *Emotion* 4 (1), 3-22

Carver, C. S., and Harmon-Jones, E. (2009) ‘Anger is an approach-related affect: Evidence and implications’. *Psychological Bulletin* 135 (2), 183–204

Carver, C. S., and White, T. L. (1994) ‘Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales’. *Journal of Personality and Social Psychology* 67 (2), 319-333

Cisler, J. M., Bacon, A. K., and Williams, N. L. (2009) ‘Phenomenological characteristics of attentional biases towards threat: a critical review’. *Cognitive Therapy and Research* 33 (1), 221-234

Cisler, J. M., and Koster, E. H. W. (2010) ‘Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review’. *Clinical Psychology Review* 30 (2), 203-216

Clark, V. P. and Hillyard, S. A. (1996) 'Spatial selective attention affects early extrastriate but not striate components of the visual evoked potential'. *Journal of Cognitive Neuroscience* 8 (5), 387-402

Clark, L. A., and Watson, D. (1991) ‘Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications’. *Journal of Abnormal Psychology* 100 (3), 316-336

Coan, J. A., Allen, J. J. B., and Harmon-Jones, E. (2001) ‘Voluntary facial expression and hemispheric asymmetry over the frontal cortex’. *Psychophysiology* 38 (6), 912–925

Coan, J. A., Allen, J. J. B., (2003) ‘Frontal EEG asymmetry and the behavioral activation and inhibition systems’. *Psychophysiology* 40 (1), 106–114

Corbetta, M., & Shulman, G. L. (2002) Control of goal-directed and stimulus-driven attention in the brain. Nature Reviews Neuroscience 3 (3), 201–15.

Crick, N. R., and Dodge, K. A. (1994) 'A review and reformulation of social-information-processing mechanisms in childrens social adjustment'. *Psychological Bulletin* 115, (1) 74-101

Cunningham, W. A., Espinet, S. D., DeYoung, C. G., and Zelazo, P. D. (2005) 'Attitudes to the right- and left: Frontal ERP asymmetries associated with stimulus valence and processing goals'. *Neuro- Image* 28 (4), 827-834

Cuthbert, B. N., Schupp, H. T., Bradley, M. M., Birbaumer, N., and Lang, P. J. (2000) 'Brain potentials in affective picture processing: Covariation with autonomic arousal and affective report'. *Biological Psychology* 52 (2), 95–111

d'Alfonso, A. A. L., van Honk, J., Hermans, E., Postma, A., and de Haan, E. H. F. (2000) 'Laterality effects in selective attention to threat after repetitive transcranial magnetic stimulation at the prefrontal cortex in female subjects'. *Neuroscience Letters* 280 (3), 195–198

Davidson, R. J. (2004) 'What does the prefrontal cortex "do" in affect: perspectives in frontal EEG asymmetry research'. *Biological Psychology* 67 (1-2), 219-234

Davidson, R. J. (2003) 'Affective neuroscience and psychophysiology: toward a synthesis'. *Psychophysiology* 40 (5), 655–665

Davidson, R. J. (1998) 'Affective style and affective disorders: Perspectives from affective neuroscience'. *Cognition & Emotion* 12 (3), 307–330

Davidson, R. J. (1995) 'Cerebral asymmetry, emotion and affective style'. in *Brain asymmetry*. ed. by R. J. Davidson and K. Hugdahl Cambridge, MA: MIT Press, 361-387

Davidson, R. J. (1994) ‘Asymmetric brain function, affective style and psychopathology: The role of early experience and plasticity’. *Development and Psychopathology 6 (4),* 741-758

Davidson, R. J. (1992a) ‘Anterior cerebral asymmetry and the nature of emotion’. *Brain and Cognition* 20 (1), 125-151

Davidson, R. J. (1992b) ‘Emotion and affective style: Hemispheric substrates’. *Psychological Science* 3 (1), 39-43

Davidson, R. J. (1984) ‘Affect, cognition, and hemispheric specialization’. in Emotions, Cognitions, and Behaviors. ed. by Izard, C. E.Kagan, J. and Zajonc, R. New York: Cambridge University Press, 320-365

Davidson, R.J., and Fox, N.A. (1989) ‘Frontal brain asymmetry predicts infants response to maternal separation’. *Journal of Abnormal Psychology* 98 (2), 127–131

Davidson, R.J., and Fox, N.A. (1982) ‘Asymmetric brain activity discriminates between positive and negative affective stimuli in 10 month old infants’. *Science* 218 (4578), 1235–1237

Davidson, R. J., and Irwin, W. (1999) ‘The functional neuroanatomy of emotion and affective style’. *Trends in Cognitive Sciences* 3 (1), 11-21

Davidson, R. J., Lewis, D., Alloy, L., Amaral, D., Bush, G., Cohen, J. Drevets, W., Farah, M., Kagan, J., McClelland, J., Nolen-Hoeksema, S., and Peterson, B. (2002) Neural and behavioural substrates of mood and mood regulation. *Biological Psychiatry* 52 (6), 478-502

Davidson, R. J., Putnam, K. M., and Larson, C. L. (2000) ‘Dysfunction in the neural circuitry of emotion regulation- A possible prelude to violence’. *Science* 289 (5479), 591–594

Davidson, R. J., and Tomarken, A. J. (1989) ‘Laterality and emotion: An electrophysiological approach’. In Handbook of neuropsychology. ed. by F. Boller and J. Grafman. Amsterdam: Elsevier, 419-441

Davis, M. (2006) 'Neural systems involved in fear and anxiety measured with fear potentiated startle'. *American Psychologist* 61 (8), 741−756

Davis, M., and Whalen, P. J. (2001) ‘The amygdala: Vigilance and emotion’. *Molecular Psychiatry* 6 (1), 13-34

Deery, S., Walsh, J., and Guest, J. (2011) 'Workplace aggression: The effects of harassment on job burnout and turnover intentions'. *Work, Employment and Society* 25 (4), 742–759

Deffenbacher, J. L. (1992) ‘Trait anger: Theory, findings, and implications’. In Advances in personality assessment. Ed. by C. D. Spielberger and J. N. Butcher. Hillsdale, NJ: Lawrence Erlbaum, 177-201

Deffenbacher, J. L., Lynch, R. S., Oetting, E. R., and Yingling, D. A. (2001) 'Driving anger: Correlates and a test of state-trait theory'. *Personality and Individual Differences* 31, 1321-1331

Dennis, T. A., and Chen, C. C. (2007) 'Emotional face processing and attention performance in three domains: Neurophysiological mechanisms and moderating effects of trait anxiety'. *International Journal of Psychophysiology* 65 (1), 10–19

Dennis, T. A., and Chen, C. C. (2009) 'Trait anxiety and conflict monitoring following threat: An ERP study'. *Psychophysiology* 46 (1), 122–131

De Raedt, R., Leyman, L., Baeken, C., Van Schuerbeek, P., Luypaert, R., Vandehasselt, M. A., et al. (2010) 'Neurocognitive effects of HF-rTMS over the dorsolateral prefrontal cortex on the attentional processing of emotional information in healthy women: an event-related fMRI study'. *Biological Psychology* 85 (3), 487–495

Derakshan, N., and Eysenck, M. W. (2009) 'Anxiety, processing efficiency, and cognitive performance new developments from attentional control theory'. *European Psychologist* 14 (2), 168–176

Derryberry. D., and Reed, M. A. (2002) 'Anxiety-related attentional biases and their regulation by attentional control'. *Journal of Abnormal Psychology* 111 (2), 225–236

de Ruiter, C., and Brosschot, J. F. (1994) 'The emotional Stroop interference in anxiety: Attentional bias or cognitive avoidance'. *Behaviour Research and Therapy* 32, 315–319

Donchin, E. (1981) 'Surprise!…. Surprise?' *Psychophysiology* 18 (5), 493–513

Donchin, E., and Coles, M. G. H. (1988) 'Is the P300 component a manifestation of context updating?' *Behaviour and Brain Sciences* 11 (3), 357–374

Donchin, E., Ritter, W., and McCallum, C. (1978) *Cognitive psychophysiology: the endogenous components of the ERP. In: Callaway P, Tueting P, Koslow S, editors. Brain-event-related potentials in man*. New York: Academic Press

Douglas, S. C., and Martinko, M. J. (2001) 'Exploring the role of individual differences in the predictions of workplace aggression'. *Journal of Applied Psychology* 86 (4), 547–559

Duncan, C. C., Barry, R. J., Connolly, J. F., Fischer, C., Michie, P. T., Naatanen, R., et al. (2009) 'Event-related potentials in clinical research: guidelines for eliciting, recording, and quantifying mismatch negativity, P300, and N400'. *Clinical Neurophysiology* 120, 1883–908

Duncan-Johnson, C. C., and Donchin, E. (1977) 'On quantifying surprise: The variation of event-related potentials with subjective probability'. *Psychophysiology* 14 (5), 456–467

Eckhardt, I. E., and Cohen, D.J. (1997) 'Attention to anger-relevant and irrelevant stimuli following naturalistic insult'. *Personality and Individual Differences* 23 (4), 619-629

Egloff, B. & Hock, M. (2003) 'Assessing attention allocation toward threat-related stimuli: a comparison of the emotional Stroop task and the attentional probe task'. *Personality and Individual Differences* 35 (2), 475-483.

Eimer, M., and Holmes, A. (2007) 'Event-related brain potential correlates of emotional face processing'. *Journal of Neurophysiology* 45 (1), 15–31

Ekman, P., and Davidson, R. J. (1993) 'Voluntary smiling changes regional brain activity'. *Psychological Science* 4(5), 342–345

Ekman, P., and Friesen, W.V. (1975) *Unmasking the face: a guide to recognizing emotions from facial clues.* Englewood Cliffs NJ: Prentice-Hall

Eldar, S., Yankelevitch, R., Lamy, D., and Bar-Haim, Y. (2010) 'Enhanced neural reactivity and selective attention to threat in anxiety'. *Biological Psychology* 85 (2), 252–257

Erwin, B. A., Heimberg, R. G., Schneider, F. R., and Liebowitz, M. R. (2003) 'Anger experience and expression in social anxiety disorder: Pre-treatment profile and

predictors of attrition and response to cognitive-behavioral treatment'. *Behavior Therapy* 34 (3), 331-350

Eysenck, M. W., and Derakshan, N. (2011) 'New perspectives in attentional control theory'. *Personality and Individual Differences* 50 (7), 955–960

Eysenck, M. W., Derakshan, N., Santos, R., and Calvo, M. G. (2007) 'Anxiety and cognitive performance: Attentional control theory'. *Emotion* 7 (2), 336−353

Eysenck, M. W., and Calvo, M. G. (1992) 'Anxiety and performance: The processing efficiency theory'. *Cognition and Emotion* 6 (6), 409–434

Falkenstein M, Hohnsbein J, Hoormann J, and Blanke L. (1991) 'Effects of crossmodal divided attention on late ERP components: II. Error processing in choice reaction tasks'. *Electroencephalogr Clin Neurophysiol* 78 (3), 447– 455.

Fava, M., and Rosenbaum, J. F. (1998) 'Anger attacks on depression'. *Depression and Anxiety* 8(S1), 59-63

Fichtenholtz, H. M., Hopfinger, J. B., Graham, R., Detwiler, J. M., and LaBar, K. S. (2007) 'Happy and fearful emotion in cues and targets modulate event-related potential indices of gaze-directed attentional orienting'. *Social Cognitive and Affective Neuroscience* 2 (4), 323–333

Fischler, I., and Bradley, M. (2006) 'Event-related potential studies of language and emotion: words, phrases, and task effects'. *Progress in Brain Research,* 156, 185–203

Fox, E. (2004) *Maintenance or capture of attention in anxiety-related biases? In: Yiend J, editor. Cognition, Emotion, and Psychopathology: Theoretical, empirical and clinical directions.* New York, New York: Cambridge University Press

Fox, N.A., and Davidson, R.J., (1986) ‘Taste-elicited changes in facial signs of emotion and the asymmetry of brain electrical activity in human newborns’. *Neuropsychologia* 24 (3), 417–422

Fox, E., Derakshan, N., and Shoker, L. (2008) 'Trait anxiety modulates the electrophysiological indices of rapid spatial orienting towards angry faces'. *Neuroreport* 19 (3), 259–263

Fox, E., Russo, R., Bowles, R., and Dutton, K. (2001) ‘Do threatening stimuli draw or hold attention in subclinical anxiety’? *Journal of Experimental Psychology, General* 130 (4), 681–700

Fox E, Russo R, and Dutton K. (2002) ‘Attentional bias for threat: Evidence for delayed disengagement from emotional faces’. *Cognition and Emotion* 16(3), 355–379

Gardener, F. L., and Moore, Z. E. (2008) ‘Understanding clinical anger and violence, the anger avoidance model’. *Behavioural modification* 32 (6), 897-912

Gainotti, G., (1972) ‘Emotional behavior and hemispheric side of the lesion’. *Cortex* 8 (3), 41–55

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., and Donchin, E. (1993) ‘A neural system for error detection and compensation’. *Psychological Science* 4 (6), 385-390

Gotlib, I. H., Ranganath, C., and Rosenfeld, J. P. (1998) ‘Frontal EEG alpha asymmetry, depression, and cognitive functioning’. *Cognition and Emotion* 12 (2), 449–478

Gray, J. A. (1990) ‘Brain systems that mediate both emotion and cognition’. *Cognition and Emotion* 4 (3), 269–288

Gray, J. A. (1987) ‘The neuropsychology of emotion and personality’. In *Cognitive neurochemistry* ed. by Stahl, S. M., Iversen, S. D. and Goodman, E. C. Oxford: Oxford University Press 171–190

Gray, J. A., and McNaughton, N. (1996) ‘The neuropsychology of anxiety: Reprise’. In: *Nebraska symposium on motivation, Perspectives on anxiety, panic, and fear. Current theory and research in motivation*, ed. by Hope D. A. Lincoln, NE: University of Nebraska Press 61-134

Gray, J. R. (2004) 'Integration of emotion and cognitive control'. *Current Directions in Psychological Science* 13 (2), 46-48

Gray, J. R., Braver, T. S., and Raichle, M. E. (2002) 'Integration of cognition and emotion in the lateral prefrontal cortex' *Proceedings of the National Academy of Sciences* 99 (6), 4115–4120

Gross, J. J. (2002) ‘Emotion regulation: Affective, cognitive, and social consequences’. *Psychophysiology* 39 (3), 281–291

Gross, J. J. (2001) ‘Emotion regulation in adulthood: Timing is everything’. *Current Directions in Psychological Science* 10 (6), 214–219

Gross, J. J. (1998) ‘Antecedent-and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology’. *Journal of Personality and Social Psychology* 74 (1), 224–237

Gross, J. J. (2007) Handbook of emotion regulation New York, NY: Guilford Press.

Gur, R.. C., Alsop, D., Glahn, D., Petty Swanson, C. L., Maldjian, J. A., Turetsky, B. I., Detre, J. A., Gee, J., and Gur, R. E. (2000) ‘An fMRI study of sex differences in regional activation to a verbal and a spatial task’. *Brain and Language* 74 (2), 157-70

Hallberg, L. R. M., and Strandmark, M. (2006) Health consequences of workplace bullying: Experiences from the perspective of employees in the public service sector. *International Methodological review of harassment research 247 Journal of Qualitative Studies on Health and Well-being* 1 (2), 109–119

Hornak, J., Bramham, J., Rolls, E. T., Morris, R.G., O’Doherty, J., Bullock, P. R., and Polkey, C. E. (2003) 'Changes in emotion after circumscribed surgical lesions or the orbitofrontal and cingulate cortices'. *Brain* 126 (7), 1691–1712

Hanoch, Y., and Vitouch, O. (2004) 'When less is more: Information, emotional arousal and the ecological reframing of the Yerkes-Dodson law'. *Theory Psychology* 14 (4), 427–452

Harmon-Jones, E. (2007) ‘Trait anger predicts relative left frontal cortical activation to anger-inducing stimuli’. *International Journal of Psychophysiology* 66 (2), 154-160

Harmon-Jones, E. (2006) ‘Unilateral right-hand contractions cause contralateral alpha power suppression and approach motivational affective experience’. *Psychophysiology* 43 (6), 598-603

Harmon-Jones, E. (2004) ‘On the relationship of anterior brain activity and anger: examining the role of attitude toward anger’. *Cognition and Emotion* 18 (1-2), 337-361

Harmon-Jones, E. (2003) ‘Anger and the behavioural approach system’. *Personality and Individual Differences* 35 (5), 995–1005

Harmon-Jones, E., and Allen, J. J. B. (1997) 'Behavioral activation sensitivity and resting frontal EEG asymmetry: covariation of putative indicators related to risk for mood disorders'. *Journal of Abnormal Psychology* 10 (6), 159–163

Harmon-Jones, E., and Allen, J. J. B., (1998) 'Anger and prefrontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence'. *Journal of Personality and Social Psychology* 74 (5), 1310–1316

Harmon-Jones, E., Gable, P. A., and Peterson, C. K. (2010) 'The role of asymmetric frontal cortical activity in emotion-related phenomena: A review and update'. *Biological Psychology* 84 (3), 451-462

Harmon-Jones, E., Lueck, L., Fearn, M., and Harmon-Jones, C., (2006) 'The effect of personal relevance and approach-related action expectation on relative left frontal cortical activity'. *Psychological Science* 17 (5), 434-440

Harmon-Jones, E., Peterson, C.K., and Harris, C.R., (2009) 'Jealousy: novel methods and neural correlates'. *Emotion* 9 113–117

Harmon-Jones, E., and Peterson, C.K. (2008) 'Effect of trait and state approach motivation on aggression'. *Journal of Research in Personality* 42 (5), 1381–1385

Harmon-Jones, E., and Sigelman, J.D. (2001) 'State anger and prefrontal brain activity: evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression'. *Journal of Personality and Social Psychology* 80 (5), 797–803

Harmon-Jones, E., Vaughn-Scott, K., Mohr, S., Sigelman, J., and Harmon-Jones, C., (2004) 'The effect of manipulated sympathy and anger on left and right frontal cortical activity'. *Emotion* 4 (1), 95–101

Harmon-Jones, E., Sigelman, J.D., Bohlig, A., and Harmon-Jones, C. (2003) ‘Anger, coping, and frontal cortical activity: the effect of coping potential on anger-induced left frontal activity’. *Cognition and Emotion* 17 (1), 1–24

Heeren, A., De Raedt, R., Koster, E. H. W., and Philippot, P. (2013) 'The (neuro)cognitive mechanisms behind attention bias modification in anxiety: proposals based on theoretical accounts of attentional bias'. *Frontiers in Human Neuroscience* 7, 119

Heeren, A., Lievens, L., and Philippot, P. (2011) 'How does attention training work in social phobia: disengagement from threat or reengagement to non-threat'? *Journal of Anxiety Disorders* 25 (4), 1108-1115

Heeren, A., Mogoase, C., McNally, R. J., Schmitz, A., and Philippot, P. (2015) 'Does attention bias modification improve attention control? A double-blind randomized experiment with individuals with social anxiety disorder'. *Journal of Anxiety Disorder* 29, 45–42

Heeren, A., Reese, H. E., McNally, R. J., and Philippot, P. (2012) 'Attention training toward and away from threat in social phobia: effects on subjective, behavioral, and physiological measures of anxiety'. *Behaviour Research and Therapy* 50 (1), 30-39.

Helfinstein, S. M., White, L. K., Bar-Haim, Y., and Fox, N. A. (2008) 'Affective primes suppress attention bias to threat in socially anxious individuals'. *Behaviour Research and Therapy* 46, 799–810.

Heller, W., and Nitschke, J. B. (1998) ‘The puzzle of regional brain activity in depression and anxiety: The importance of subtypes and comorbidity’. *Cognition and Emotion* 12 (3), 421–447

Henriques, J.B., and Davidson, R.J. (1990) ‘Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects’. *Journal of Abnormal Psychology* 99 (1), 22–31

Herbert, C., Junghofer, M., and Kissler, J. (2008) 'Event-related potentials to emotional adjectives during reading'. *Psychophysiology* 45 (3), 487-498

Herbert, C., KisslerHerbert, C., Kissler, J., Junghfer, M., Peyk, P., and Rockstroh, B. (2006) 'Processing of emotional adjectives: evidence from startle EMG and ERPs'. *Psychophysiology* 43 (2), 197–206

Hewig J, Hagemann D, Seifert J, Naumann E, and Bartussek D. (2006) ‘The relation of cortical activity and BIS/BAS on the trait level’. *Biological Psychology* 71(1), 42–53.

Herwig, U., Padberg, F., Unger, J., Spitzer, M., and Schonfeld-Lecuona, C. (2001) ‘Transcranial magnetic stimulation in therapy studies: examination of the reliability of “standard” coil positioning by neuronavigation’. *Biological Psychiatry* 50, 58–61

Hewig, J., Hagemann, D., Seifert, J., Naumann, E., Bartussek, D. (2004) ‘On the selective relation of frontal cortical activity and anger-out versus anger-control’. *Journal of Personality and Social Psychology* 87 (6), 926–939

Herwig, U., Satrapi, P., & Schonfeldt-Lecuona, C. (2003) ‘Using the international 10–20 EEG system for positioning of transcranial magnetic stimulation’. *Brain Topography* 16, 95–99

Hillyard, S. A., Mangun, G. R.,Woldor, M. G. & Luck, S. J. 1995 *Neural systems mediating selective attention. In The cognitive neurosciences (ed. M. S. Gazzaniga).* Cambridge MA: MIT Press.

Hillyard, S. A., and Vento, L. A. (1998) 'Event-related brain potentials in the study of visual selective attention' *Proceedings of the National Academy of Science USA* 95 (3), 781–887

Hornak, J., Bramham, J., Rolls, E. T., Morris, R. G., O'Doherty, J., Bullock, P. R., and Polkey, C. E. (2003) 'Changes in emotion after circumscribed surgical lesions or the orbitofrontal and cingulate cortices'. *Brain* 126 (7), 1691–1712

Ille, N., Berg, P., and Scherg, M. (2002) 'Artifact correction of the ongoing EEG using spatial filters based on artifact and brain signal topographies'. *Clinical Neurophysiology* 19 (2), 113–124

Jennings, J. R., and Wood, C. C. (1976) 'The e-adjustment procedure for repeated-measures analyses of variance'. *Psychophysiology* 13 (3), 277–278

Johnson, R., Jr. (1986) 'A triarchic model of P300 amplitude'. *Psychophysiology* 23 (4), 367–384

Johnston, V. S., Miller, D. R., and Burleson, M. H. (1986) 'Multiple P3s to emotional stimuli and their theoretical significance'. *Psychophysiology* 23 (6), 684–694

Jones, A. D., Cho, R. Y., Nystrom, L. E., Cohen, J. D., and Braver, T. S. (2002) 'A computational model of anterior cingulate function in speeded response tasks: Effects of frequency, sequence, and conflict'. *Cognitive, Affective & Behavioral Neuroscience* 2 (4), 300–317

Kanske, P., and Kotz, S. A. (2007) 'Concreteness in emotional words: ERP evidence from a hemifield study'. *Brain Research* 1148, 138–148

Kawasaki, H., Adolphs, R., Kaufman, O., Damasio, H., Damasio, A. R., Granner, M., Bakken, H., Hori, T., and Howard, M. A. III (2001) ‘Single-neuron responses to emotional visual stimuli recorded in human ventral prefrontal cortex’. *Nature Neuroscience* 4 (1), 15–16

Kenemans, J. L., Kok, A., and Smulders, F. T. Y. (1993) 'Event-related potentials to conjunctions of spatial frequency and orientation as a function of stimulus parameters and response requirements'. *Electroencephalograph Clinical Neurophysiology* 88 (1), 1 – 63

Kissler, J., Herbert, C., Winkler, I., and Junghöfer, M. (2009) 'Emotion and attention in visual word processing: An ERP study'. *Biological Psychology* 80 (1), 75-83

Konishi, S., Nakajima, K., Uchida, I., Kikyo, H., Kameyama, M., and Miyashita, Y.(1999) 'Common inhibitory mechanism in human inferior prefrontal cortex revealed by event-related functional MRI'. *Brain* 122 (5), 981–991

Koster, E. H. W., Crombez, G., Vershuere, B., Van Damme, S., and Wiersema, J. R. (2006) 'Components of attentional bias to threat in high trait anxiety: Facilitated engagement, impaired disengagement, and attentional avoidance'. *Behaviour Research and Therapy* 44 (12), 1757−1771

Koster, E. H., Crombez, G., Verschuere, B., and De Houwer, J. (2004) ‘Selective attention to threat in the dot probe paradigm: Differentiating vigilance and difficulty to disengage’. *Behaviour Research and Therapy* 42 (10), 1183–1192

Koster, E. H. W., Verschuere, B., Crombez, G., and Van Damme, S. (2005) 'Time-course of attention for threatening pictures in high and low trait anxiety'. *Behaviour Research and Therapy* 43 (8), 1087−1098

Koster, E. H., Crombez, G., Verschuere, B., and De Houwer, J. (2006) ‘Attention to threat in anxiety-prone individuals: Mechanisms underlying attentional bias’. *Cognitive Therapy and Research* 30 (5), 635−643

Kring, A. M., and Bachorowski, J. A. (1999) 'Emotions and psychopathology'. *Cognition and Emotion* 13 (5), 575−599

Lang, P. J., Bradley, M. M., and Cuthbert, B. N. (1990) 'Emotion, attention, and the startle reflex'. *Psychological Review* 97 (12), 377–95

Lazarus, R. S. (1991) *Emotion and adaptation*. New York: Oxford

Lavie, N. (1995) 'Perceptual load as a necessary condition for selective attention'. *Journal of Experimental Psychology: Human Perception and Performance* 21 (3), 451–468

Lavie, N., Hirst, A., de Fockert, J., and Viding, E. (2004) 'Load theory of selective attention and cognitive control'. *Journal of Experimental Psychology*: *General* 133 (3), 339-354

LeDoux, J. E. (2000) 'Emotion circuits in the brain'. *Annual Review of Neuroscience* 23, 155−184.

LeDoux, J. E. (1996) *The emotional brain*. New York: Simon & Schuster

Leyman, L., De Raedt, R., Vanderhasselt, M. A., and Baeken, C. (2009) 'Influence of high-frequency repetitive transcranial magnetic stimulation over the dorsolateral prefrontal cortex on the inhibition of emotional information in healthy volunteers'. *Psychol. Med* 39 (6), 1019–1028

Li, W., Zinbarg, R. E., Paller, K. A. (2007) 'Trait anxiety modulates supraliminal and subliminal threat: Brain potential evidence for early and late processing influences'. *Cognitive, Affective, & Behavioural Neuroscience* 7 (1), 25–36

Liotti, M., Woldorff, M. G., Perez, R., and Mayberg, H. S. (2000) 'An ERP study of the temporal course of the Stroop color–word interference effect'. *Neuropsychologia* 38 (5), 701–711

Litt, M. D., Cooney, N. L., and Morse, P. (2000) 'Reactivity to alcohol-related stimuli in the laboratory and in the field: predictors of craving in treated alcoholics'. *Addiction* 95 (6), 889–900

Luck, S. J. (2005) *An Introduction to the Event-Related Potential Technique*. Cambridge, MA: MIT Press

Luck, S. J., Heinze, H. J., Mangun, G. R., and Hillyard, S. A. (1990) 'Visual event-related potentials index focused attention within bilateral stimulusarrays. II. Functional dissociation of P1 and N1 components'. *Electroencephalography and Clinical Neurophysiology* 75 (6), 528–542

MacDonald, A.W., Cohen, J.D., Stenger, V.A., and Carter, C.S. (2000) 'Dissociating the role of dorsolateral prefrontal cortex and anterior cingulate cortex in cognitive control'. *Science* 288 (5472) 1835–38

MacLeod, C., and Hagan, R. (1992) 'Individual differences in the selective processing of threatening information, and emotional responses a stressful life event'. *Behaviour Research and Therapy* 30 (2), 151-161

Macleod, C., and Mathews, A. (2012) 'Cognitive bias modification approaches to anxiety' *Annual Review of Clinical Psychology* 8, 189-217

MacLeod, C., Mathews, A., and Tata, P. (1986) ‘Attentional bias in emotional disorders’. *Journal of Abnormal Psychology* 95 (1), 15-20

MacNamara, A., Foti, D., & Hajcak, G. (2009) Tell me about it: Neural activity elicited by emotional stimuli and preceding descriptions. *Emotion* 9 (4), 531-543.

Mangun, G. R., and Hillyard, S. A. (1995) *Mechanisms and models of selective attention. In: Rugg,M.D., Coles, M.G.H. ŽEds.., Electrophysiology of Mind.* Oxford: Oxford University Press

Mather, M., and Sutherland, M. (2009) ‘Disentangling the effects of arousal and valence on memory for intrinsic details’. *Emotion Review* 1 (2), 118-119

Mather, M., and Sutherland, M. (2011) ‘Arousal-biased competition in perception and memory’. *Perspectives on Psychological Science* 6 (2), 114-133

Markela-Lerenc, J., Ille, N., Kaiser, S., Fiedler, P., Mundt, C., and Weisbord, M. (2004) 'Prefrontal-cingulate activation during executive control: which comes first'? *Cognitive Brain Research* 18 (1), 278–287

Mathersul D, Williams L. M., Hopkinson P. J., and Kemp A.H. (2008) ‘Investigating models of affect: Relationships among EEG alpha asymmetry, depression, and anxiety’. *Emotion* 8 (4), 560–572

Mathews, A., and Mackintosh, B. (1998) ‘A cognitive model of selective processing in anxiety’. *Cognitive Therapy and Research* 22 (6), 539-560

Mathews, A., and MacLeod, C. (2002) 'Induced processing biases have causal effects on anxiety'. *Cognition and Emotion* 16 (3), 331–54

Mathews, A., Mogg, K., Kentish, J., & Eysenck, M. (1995) 'Effect of psychological treatment on cognitive bias in generalized anxiety disorder'. *Behavior Research and Therapy* 33 (3), 293–303

Mathews, A., and MacLeod, C. (1994) 'Cognitive approaches to emotion and emotional disorders'. *Annual Review of Psychology* 45, 25-50

Matthews, G., & Wells, A. (2000) 'Attention, automaticity, and affective disorder'. *Behavior Modification* 24 (1), 69−93.

Mauss, I. B., Evers, C., Wilhelm, F. H., and Gross, J. J. (2006) 'How to bite your tongue without blowing your top: Implicit evaluation of emotion regulation predicts affective responding to anger provocation'. *Personality and Social Psychology Bulletin* 32 (5), 589-602

McNally, R. J. (1995) 'Automaticity and the anxiety disorders'. *Behaviour Research and Therapy* 33 (7), 747−754

Meinhardt, J., and Pekrun, R. (2003) 'Attentional resource allocation to emotional events: An ERP study'. *Cognition and Emotion* 17 (3), 477–500

Miller, E. K., and Cohen, J. D. (2001) 'An integrative theory of prefrontal cortex function'. *Annual Review of Neuroscience* 24, 167-202

Mitchell, I., and Gilchrist, E. (2006) 'Domestic violence and panic attacks – common neural mechanisms'? *Legal and Criminological Psychology* 11(2), 267-282

Mogg, K., Bradley, B. P., De Bono, J., and Painter, M. (1997) 'Time course of attentional bias for threat information in non-clinical anxiety'. *Behaviour Research and Therapy* 35 (4), 297-303

Mogg, K., and Bradley, B. P. (2005) 'Attentional bias in generalized anxiety disorder versus depressive disorder'. *Cognitive Therapy and Research* 29 (1), 29–45

Mogg, K., Bradley, B. P., Dixon, C., Fisher, S., Twelftree, H., and McWilliams, A. (2000) 'Trait anxiety, defensiveness and selective processing of threat: an investigation using two measures of attentional bias'. *Personality and Individual Differences* 28 (6), 1063–1077

Mogg, K., Bradley, B. P., Miles, F., and Dixon, C. (2004) 'Time course of attentional bias for threat scenes: Testing the vigilance-avoidance hypothesis'. *Cognition and Emotion* 18 (5), 689−700

Mogg, K., and Bradley, B. P. (2002) 'Selective orienting of attention to masked threat faces in social anxiety'. *Behaviour Research and Therapy* 40 (12), 1403-1414

Mogg, K., and Bradley, B. P. (1998) 'A cognitive-motivational analysis of anxiety'. *Behaviour Research and Therapy* 36 (9), 809–848

Mogg, K., Bradley, B. P., Dixon, C., Fisher, S., Twelftree, H., and McWilliams, A. (2000) 'Trait anxiety, defensiveness and selective processing of threat: an investigation using two measures of attentional bias'. *Personality and Individual Differences* 28 (6), 1063-1077

Mogg, K., Bradley, B. P., Williams, R., & Mathews, A. (1993) 'Subliminal processing of emotional information in anxiety and depression'. *Journal of Abnormal Psychology* 102 (2), 304−311

Moors, A., & De Houwer, J. (2006) 'Automaticity: A theoretical and conceptual analysis'. *Psychological Bulletin* 132 (2), 297−326

Mueller, E. M., Hofmann, S. G., Santesso, D. L., Meuret, A. E., Bitran, S., and Pizzagalli, D. A. (2009) 'Electrophysiological evidence of attentional biases in social anxiety disorder'. *Psychological Medicine* 39 (7), 1141–1152

Murphy, R. T., Cameron, R. P., Sharp, L., Ramirez, G., Rosen, C. S., and Drescher, K. (2004) 'Readiness to change PTSD symptoms and other problems among veterans participating in a motivation enhancement group'. *The Behaviour Therapist* 27 (2), 33-36

Nieuwenhuis, S., Aston-Jones, G., and Cohen, J. D. (2005) 'Decision making, the P3, and the locus coeruleus--norepinephrine system'. *Psychological Bulletin* 131 (4), 510–532

Nieuwenhuis, S., Yeung, N., Van Den Wildenberg, W,. and Ridderinkhof K. R. (2003) 'Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency'. *Cognitive, Affective & Behavioral Neuroscience* 3 (1), 17–26

O'Doherty J, Kringelbach M.L., Rolls E.T., Hornak J., and Andrews C. (2001) 'Abstract reward and punishment representations in the human orbitofrontal cortex'. *Nature Neuroscience* 4, 95-102

Öhman A. (2005) 'The role of the amygdala in human fear: automatic detection of threat'. *Psychoneuroendocrinology* 30 (10), 953–958

Öhman, A. (1996) 'Preferential preattentive processing of threat in anxiety: preparedness and attentional biases'. In *Current Controversies in the Anxiety Disorders*. Ed. by Rapee R. M. New York: Guilford Press 253–290

Öhman, A., and Mineka, S. (2001) 'Fear, phobias and preparedness: Toward an evolved module of fear and fear learning'. *Psychological Review* 108 (3), 483-522

Oldfield, R.C. (1971) ‘The assessment and analysis of handedness: The Edinburgh inventory’. *Neuropsychologia* 9 (1), 97–113

O’Toole, L. J., & Dennis, T. A. (2012) Attention training and the threat bias: An ERP study. *Brain and Cognition* 78 (1), 63–73.

Paulhus, D. L., and Vazire, S. (2007) ‘The self-report method’. In *Handbook of Research Methods in Personality Psychology*. Ed. by Robins, R. W., Fraley, R. C., and Krueger, R. New York: Guilford Press 224-239

Penfield, W. (1958) ‘Some mechanisms of consciousness discovered during electrical stimulation of the brain’. *Proceedings of the National Academy of Sciences, U.S.A.,* 44, 51–66.

Peterson, C.K., Shackman, A.J., and Harmon-Jones, E., (2008) ‘The role of asymmetric frontal cortical activity in aggression’. *Psychophysiology* 45 (1), 86–92

Perez-Edgar, K., Fox, N.A., 2003. Individual differences in children's performance during an emotional Stroop task: a behavioral and electrophysiological study. Brain and Cognition 52 (1), 33–51.

Perez-Edgar, K., Kajawa, A., Nelson, S.K., Cole, C., and Zapp, D.J.,(2013) ‘The relation between electroencephalography asymmetry and attention biases to threat at baseline and under stress’. *Brain and Cognition* 82 (3), 337-343

Phaf, R. H., and Kan, K. J. (2007) ‘The automaticity of emotional Stroop: A meta-analysis’. *Journal of Behaviour Therapy and Experimental Psychiatry* 38 (,184–199

Phan, K. L., Fitzgerald, D. A., Nathan, P. J., and Tancer, M. E. (2006) 'Association between amygdala hyperactivity to harsh faces and severity of social anxiety in generalized social phobia'. *Biol. Psychiatry* 59 (5), 424–429

Phelps, E. A., and LeDoux, J. E. (2005) 'Contributions of the amygdala to emotion processing: From animal models to human behavior'. *Neuron* 48 (2), 175–187

Phillips, M. L., Ladouceur, C. D., and Drevets, W. C. (2008) 'A neural model of voluntary and automatic emotion regulation: Implications for understanding the pathophysiology and neurodevelopment of bipolar disorder'. *Molecular Psychiatry* 13 (9), 833–857

Pishyar, R., Harris, L. M., and Menzies, R. G. (2004) 'Attentional bias for words and faces in social anxiety'. *Anxiety, Stress, and Coping* 17 (1), 23–36

Plutchik, R.(1980) *Emotion: A Psychoevolutionary Synthesis*. New York, NY: Harpercollins College Division

Polich, J. (2007) 'Updating P300: An integrative theory of P3a and P3b'. *Clinical Neurophysiology* 118 (10), 2128–2148

Posner, M. I. (1980) 'Orienting of attention'. *Quarterly Journal of Experimental Psychology* 32 (1), 3–25

Posner, M .I., and Petersen, S. E. (1990) 'The attention system of the human brain'. *Annu. Rev. Neurosc* 13, 25– 42

Posner, M. R., and Rothbart, M. K. (2000) 'Developing mechanisms of self-regulation'. *Development and Psychopathology* 12 (2), 427−441

Posner, M. I., Snyder, C. R. and Davidson, B. J. (1980) 'Attention and the detection of signals'. *Journal of Experimental Psychology* 109 (2), 160–74

Potts, G. F., and Tucker, D. M. (2001) 'Frontal evaluation and posterior representation in target detection'. *Brain Res. Cogn. Brain Res.* 11 (1), 147– 156

Pury, C.L.S. (2002) 'Information-processing predictors of emotional response to stress'. *Cognition and Emotion* 16(5), 667-683

Putman, P., Arias-Garcia, E., Pantazi, I., and Van Schie, C. (2012) 'Emotional Stroop interference for threatening words is related to reduced EEG delta–beta coupling and low attentional control'. *International Journal of Psychophysiology* 84 (2), 194–200

Putman, P., Hermans, E., and van Honk, J. (2004) 'Emotional stroop performance for masked angry Faces: It's BAS, Not BIS'. *Emotion* 4 (3), 305-311

Robinson, R.G., and Price, T.R., (1982) 'Post-stroke depressive disorders: a follow-up study of 103 patients'. *Stroke* 13 (5), 635–641

Roelofs, K., Putman, P., Schouten, S., Lange, W.-G., Volman, I., & Rinck, M. (2010) Gaze direction differentially affects avoidance tendencies to happy and angry faces in socially anxious individuals. *Behaviour Research and Therapy.* 48 (4), 290–294.

Rolls, E.T., Kringelbach, M.L., and de Araujo, I.E., (2003) 'Different representations of pleasant and unpleasant odours in the human brain'. *European Journal of Neuroscience* 18 (3), 695–703

Rolls, E .T. and Grabenhorst, F. (2008) 'The orbitofrontal cortex and beyond: from affect to decision-making'. *Progress in Neurobiology* 86 (3), 216-244

Rolls, E.T., (1999) *The Brain and Emotion*. New York: Oxford University Press

Rossi, S., Hallett, M., Rossini, P. M., and Pascual-Leone, A. (2009) 'The safety of TMS consensus group. Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research'.*Clin. Nurophysiol* 120 (12), 2008–2039

Rugg, M. D., and Coles, M. G. H. (1995) *Electrophysiology of mind: event-related brain potentials and cognition*. Oxford: Oxford University Press

Sack A. T., Cohen Kadosh R., Schuhmann, T., Moerel M., Walsh, V., and Goebel, R. (2009) 'Optimizing functional accuracy of TMS in cognitive studies: a comparison of methods'. *J Cogn Neurosci*. 21, 207–21

Sackeim, H., Greenberg, M.S., Weimen, A.L., Gur, R.C., Hungerbuhler, J.P., and Geschwind, N. (1982) 'Hemispheric asymmetry in the expression of positive and negative emotions: neurologic evidence'. *Archives of Neurology* 39, 210–218

Schapkin, S.A., Gusev, A.N., Kuhl, J., 2000. Categorization of unilaterally presented emotional words: an ERP analysis. Acta Neurobiol. Exp. 60 (1), 17–28.

Scherg, M. and Berg, P. (1996) *BESA-Brain Electric Source Analysis Handbook, Max-Planck Institute for Psychiatry*. Munich:

Schiff, B.B., and Lamon, M., (1994) 'Inducing emotion by unilateral contraction of hand muscles'. *Cortex* 30 (2), 247–254

Schiff, B.B., and Truchon, C. (1993) 'Effect of unilateral contraction of hand muscles on perceiver biases in the perception of chimeric and neutral faces' *Neuropsychologio* 31 (12) 1351-1365

Schmidt, N. B., Richey, J. A., Buckner, J. D., and Timpano, K. R. (2009) 'Attention training for generalized social anxiety disorder'. *Journal of Abnormal Psychology* 118 (1), 5-14

Schutter, Van Honk, d' Alfonso, Postma, and De Haan, (2001) 'Effects of slow rTMS at the right dorsolateral prefrontal cortex on EEG asymmetry and mood'. *NeuroReport* 12 (3), 445–447

Sekihara, K., Nagarajan, S., Poeppel, D., and Marantz, A. (2002) 'Performance of an MEG adaptive-beamformer technique in the presence of correlated neural activities: Effects on signal intensity and time-course estimates'. *IEEE Trans Biomedical Engineering* 40 (10), 1534–1546.

Shiffrin, R. M., and Schneider, W. (1977) 'Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory'. *Psychological Review* 84 (2), 127−190

Siemer, M., Mauss, I., and Gross, J. J. (2007) 'Same situation– different emotions: How appraisals shape our emotions'. *Emotion* 7 (3), 592–600

Sommer, I. E.C., Aleman, A., Bouma, A, and Kahn R. S. (2004) 'Do women really have more bilateral language representation than men? A meta-analysis of functional imaging studies'. *Brain* 127, 1845-1852

Smith, T. W., Glazer, K., Ruiz, J. M., and Gallo, L. C. (2004) 'Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion and health'. *Journal of Personality* 72 (6), 1217–1270

Smith, P., and Waterman, M.G. (2003) 'Processing bias for aggression words in forensic and non-forensic samples'. *Cognition and Emotion* 17 (5), 681–701

Smith, P., and Waterman, M. G. (2004) 'Processing Bias for Sexual Material: The Emotional Stroop and Sexual Offenders'. *Sexual Abuse: A Journal of Research and Treatment* 16 (2), 163-171

Smith, P., and Waterman, M.G. (2005) 'Sex differences in processing aggression words using the Emotional Stroop task'. *Aggressive Behavior* 31 (3), 271-282

Smits, D.J.M., and Kuppens, P., (2005) 'The relations between anger, coping with anger, and aggression, and the BIS/BAS system'. *Personality and Individual Differences* 39 (4), 783–793

Spielberger, C. D. (1999) *State Trait Anger Expression Inventory-2*. Lutz, Florida: Psychological Assessment Resources

Spielberger, C. D., Foreyt, J. P., Goodrick, G. K., & Reheiser, E. C. (1995) Personality characteristics of user of smokeless tobacco compared with cigarette smokers and non-users of tobacco products. *Personality and Individual Differences* 19 (4), 439-448.

Stein, M. B., Goldin, P. R., Sareen, J., Zorrilla, L. T. E., and Brown, G. G. (2002) Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Arch. Gen. Psychiatry* 59 (11), 1027–1034

Stein, M.B., Simmons, A.N., Feinstein, J.S., and Paulus, M.P. (2007) 'Increased amygdala and insula activation during emotion processing in anxiety-prone subjects' *Am. J. Psychiatry* 164 (2), 318–327

Stewart, J. L., Levin Silton, R., Sass, S. M., Fisher, J. E., Edgar, J. C., Heller, W., and Miller, G. A. (2010) 'Attentional bias to negative emotion as a function of approach and withdrawal anger styles: an ERP investigation'. *Int. J. Psychophysiol* 76 (1), 9–18

Sutton, S. K., and Davidson, R. J. (1997) ‘Prefrontal Brain Asymmetry: A Biological Substrate of the Behavioral Approach and Inhibition Systems’. *Psychological Science* 8 (3), 204-210

Taake, I., Jaspers-Fayer, F., and Liotti, M. (2009) 'Early frontal responses elicited by physical threat words in an emotional Stroop task: Modulation by anxiety sensitivity'. *Biological Psychology* 81(1), 48–57

Thatcher, R. W., Krause, P. J., & Hrybyk, M. (1986) ‘Cortico-cortical associations and EEG coherence: A two-compartmental model’. *Electroencephalography and Clinical Neurophysiology*, 64, 123–143

Thomas, S.J., Johnstone, S.J., Gonsalvez, C.J., (2007) Event-related potentials during an emotional Stroop task. *International Journal of Psychophysiology* 63 (3), 221–231

Tomarken, A.J., Davidson, R.J., Wheeler, R.E., and Doss, R. (1992) ‘Individual differences in anterior brain asymmetry and fundamental dimensions of emotion’. *Journal of Personality and Social Psychology* 62 (4), 676–687

Tomarken, A.J., Davidson, R.J., and Henriques, J.B., (1990) ‘Resting frontal brain asymmetry predicts affective responses to films’. *Journal of Personality and Social Psychology* 59 (4), 791–801

Todorov, A., and Bargh, J. A. (2002) 'Automatic sources of aggression'. *Aggression and Violent Behavior* 7, 53–68

Tranel, D., Bechara, A., and Denburg, N.L., (2002) ‘Asymmetric functional roles of right and left ventromedial prefrontal cortices in social conduct, decision-making and emotional processing’ *Cortex* 38 (4), 589–612

Trauer S. M., Kotz S. A., and Müller M. M. (2015) ‘Emotional words facilitate lexical but not early visual processing’ *BMC Neurosci* 16, 1-9

Usakli, A. B. (2010) ‘Improvement of EEG signal acquisition: An electrical aspect for state of the art of front end’. *Computational Intelligence and Neuroscience* 2010, 630649

van Honk, J., and Schutter, D.J.L.G. (2006) ‘From affective valence to motivational direction: the frontal asymmetry of emotion revisited’. *Psychological Science* 17 (11), 963–965

van Honk, J., Tuiten, A., van den Hout, M., Putnam, P., de Haan, E., and Stam, H. (2001) ‘Selective attention to unmasked and masked threatening words: relationships to trait anger and anxiety’. *Personality and Individual Differences* 30 (4), 711–720

van Honk, J., Tuiten, A., de Haan, E., van den Hout, M., and Stam, H. (2001) ‘Attentional biases for angry faces: relationship to trait anger and anxiety’. *Cognition and Emotion* 15, 279–297

van Honk, J., Schutter,D. J. L. G., d’Alfonso, A.A. L., Kessels, R. P. C., and de Haan, E. H. F. (2002) ‘1 hz rTMS over the right prefrontal cortex reduces vigilant attention to unmasked but not to masked fearful faces’. *Biological Psychiatry* 52 (4), 312- 317

Van Hooff, J.C., Dietz, K.C., Sharma, D., and Bowman, H. (2008) 'Neural correlates of intrusion of emotion words in a modified Stroop task'. *International Journal of Psychophysiology* 67, 23–34

Van Petten, C., and Kutas, M. (1990) 'Interactions between sentence context and word frequency in event-related brain potentials'. *Memory & Cognition* 18 (4), 380–393

Van Veen, V., Carter, C. S. (2002) 'The anterior cingulate as a conflict monitor: fMRI and ERP studies'. *Physiology & Behavior* 77 (4-5), 477–482

Wallis, J.D., and Miller, E.K., (2003) 'Neuronal activity in primate dorsolateral and orbital prefrontal cortex during performance of a reward preference task'. *European Journal of Neuroscience* 18 (7), 2069–2081

Watson, D. (2009) 'Locating anger in the hierarchical structure of affect: Comment on Carver and Harmon-Jones (2009)'. *Psychological Bulletin* 135 (2), 205–208

Watson, D. (2000) *Mood and Temperament*. New York: Guilford Press

Wells, A., & Matthews, G. (1994) Attention and emotion: A clinical perspective. Hillsdale, NJ: Lawrence Erlbaum Associates.

West, R., 2003. Neural correlates of cognitive control and conflict detection in the Stroop and digit-location tasks. Neuropsychologia 41, 1122–1135.

West, R., and Alain, C. (2000) 'Effects of task context and fluctuations of attention on neural activity supporting performance of the Stroop task'. *Brain Res*. 873 (1), 102–111.

West, R., and Alain, C. (1999) 'Event-related neural activity associated with the Stroop task'. *Cogn. Brain Res*. 8 (2), 157–164

Wilkowski, B., and Robinson, M. (2008) 'The cognitive basis of trait anger and reactive aggression: An integrative analysis'. *Personality and Social Psychology Review* 12 (1), 3–21

Wilkowski, B. M., Robinson, M. D., & Meier, B. P. (2006) Agreeableness and the prolonged spatial processing of antisocial and prosocial information. *Journal of Research in Personality* 40 (6), 1152-1168.

Williams, S. M. (1986) ‘Factor Analysis of the Edinburgh Handedness Inventory’. *Cortex*, 22 (2), 325-326

Williams, J. M., Mathews, A., and MacLeod, C. M., (1996) ‘The emotional Stroop task and psychopathology’. *Psychological Bulletin* 120 (1), 3-24

Williams, J. E. , Nieto,F. J. , Sanford, C. P. Couper, D. J. and Tyroler H. A. (2002) ‘The Association Between Trait Anger and Incident Stroke Risk: The Atherosclerosis Risk in Communities (ARIC) Study’. *Stroke* 33 (1), 13-20

Williams, J. E., Paton, C. C., Siegler, I. C., Eigenbrodt, M. L., Nieto, F. J. and Tyroler H. A. (2000) ‘Anger Proneness Predicts Coronary Heart Disease Risk: Prospective Analysis From the Atherosclerosis Risk In Communities (ARIC) Study’. *Circulation* 101 (17), 2034-2039

Williams, J. M. G., Watts, F. N., MacLeod, C., and Mathews, A. (1988) *Cognitive psychology and emotional disorders*. Chichester: Wiley

Williams, J. M.G., Watts, F.N., MacLeod, C., and Mathews, A. (1997) *Cognitive Psychology and Emotional Disorders*. II edition. Chichester: Wiley

Wheeler, R.E., Davidson, R.J., and Tomarken, A.J., (1993) ‘Frontal brain asymmetry and emotional reactivity: a biological substrate of affective style’. *Psychophysiology* 30 (1), 82–89

Yiend, J., and Mathews, A. (2001) ‘Anxiety and attention to threatening pictures’. *Quarterly Journal of Experimental Psychology Section a-Human Experimental Psychology* 54 (3), 665-681

Appendix 1 EST Word List

Threat words		Neutral words	
Accident	Exam	Hat	Bricklayer
Afraid	Fight	Add	Brush
Ambulance	Fail	Bag	Keyboard
Amputation	Fear	Cable	Form
Angry	Force	Beard	Cement
Assault	Fire	Ear	Cotton
Attack	Flood	Poem	Track
Death	Harassed	Walk	Pavement
Bleed	Hurricane	Nose	February
Burn	Hurt	Look	Paint
Break	Knife	Letter	Similar
Beaten	Kill	Floor	Message
Brutal	Needle	Smooth	Next
Cancer	Pain	Paintbrush	Liquid
Crime	Poison	Cheque	Plate
Combat	Punish	Bronze	Button
Crash	Paralysed	Cardboard	Leaf
Criticism	Operation	Shoe	Identical
Cry	Offended	Light	Similar
Dangerous	Rape	Bird	House
Destroy	Savage	Shoulder	Ceiling
Dead	Stabbed	Tent	Structure
Despair	Stupid	Close	Curtain
Disaster	Surgery	Bridge	Ankles
Disease	Suffocate	Harbour	Booklet
Drowned	Tornado	Model	Cereal
Earthquake	Torture	Path	Farmer
Emergency	Virus	Morning	Metal
Explosion	Murder	Mountain	Wings
Enemy	Weapon	Approach	Anchor

Appendix 2 DPT Word List

Threat words		
Lie	Drown	Crucify
Sin	Devil	Destroy
War	Fraud	Crushed
Die	Scorn	Despise
Cry	Thief	Divorce
Hit	Ulcer	Enraged
Bomb	Upset	Selfish
Debt	Roach	Fearful
Burn	Venom	Hostage
Dead	Misery	Hostile
Hate	Cancer	Violent
Hell	Brutal	Suicide
Hurt	Insult	Mistake
Jail	Danger	Penalty
Lice	Burial	Pervert
Lost	Coffin	Torture
Pain	Crisis	Bankrupt
Rape	Killer	Burdened
Riot	Deceit	Defeated
Ugly	Detest	Helpless
Rude	Guilty	Disaster
Abuse	Morbid	Disloyal
Agony	Maggot	Murderer
Crash	Malice	Sickness
Anger	Menace	Dreadful
Angry	Stress	Headache
Annoy	Sinful	Intruder
Crime	Prison	Jealousy
Slave	Putrid	Mutilate
Cruel	Rabies	
Demon	Robber	
Toxic	Scared	
Filth	Assault	

Positive words		
Ace	Charm	Admired
Car	Youth	Inspire
Eat	Flirt	Aroused
Fun	Heart	Ecstasy
Sex	Jolly	Sunrise
God	Greet	Engaged
Baby	Loyal	Gymnast
Awed	Loved	Victory
Bake	Smile	Hopeful
Bold	Circus	Magical
Cake	Desire	Justice
Cash	Dinner	Improve
Cute	Comedy	Holiday
Face	Bright	Miracle
Foam	Famous	Passion
Food	Couple	Wealthy
Rage	Joyful	Ambition
Free	Dancer	Laughter
Good	Casino	Paradise
Game	Caress	Optimism
Gift	Father	Applause
Alert	Erotic	Powerful
Alive	Elated	Birthday
Event	Dollar	Pleasure
Beach	Joyful	Intimate
Brave	Nipple	Sunlight
Glory	Chance	Terrific
Cheer	Heaven	Inspired
Money	Garter	Positive
Happy	Friend	
Blond	Kitten	
Bride	Honest	
Champ	Lottery	

Neutral words		
Arm	Coast	Cabinet
Egg	Diver	Context
Cow	Phase	Hairpin
Hat	Plant	Nursery
Hey	Quart	Journal
Ink	Salad	Passage
Bowl	Spray	Ketchup
Chin	Paper	Privacy
Cord	Quiet	Whistle
Cork	Kettle	Utensil
Door	Limber	Machine
Farm	Butter	Trumpet
Foot	Poetry	Patient
Fork	Street	Prairie
Frog	Museum	Village
Hand	Fabric	Teacher
Hawk	Circle	Industry
Step	Office	Bathroom
Tape	Statue	Building
Path	Golfer	Material
Item	Gender	Kerchief
Ankle	Theory	Umbrella
Watch	Method	Consoled
Chair	Modest	Medicine
Table	Window	Scissors
Clock	Yellow	Mushroom
Elbow	Pencil	Reverent
Horse	Patent	Windmill
Jelly	Poster	Activate
Month	Sphere	
Tower	Rattle	
Paint	Violin	
Black	History	